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THE PERIVASCULAR NEURAL PATTERN OF THE FEMORAL REGION

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THIS account concerns the anatomy of the 'nerves to blood-vessels', a problem that has excited the attention of anatomists and surgeons for many years. The classic nineteenth-century anatomists were well aware of the existence of branches from certain of the peripheral nerves to the main limb arteries, and their accounts of the topography of such nerves have never been excelled.

The present investigation was undertaken as an attempt to bridge the gap between these gross macroscopical appearances, and those microscopical features of blood-vessel innervation concerning which the literature affords many exhaustive accounts. Human fœtuses preserved in a particular manner, and dissected under the low-power microscope, show certain topographical differences between (*a*) the neural plexus upon main axial blood-vessels and (*b*) the long and exceedingly delicate nerve filaments supplying deep arteries to skeletal muscle, and other collateral vessels.

The conclusions deriving from this topographical study are discussed in relation to present-day concepts concerning the morbid physiology and treatment of intermittent claudication.

HISTORICAL INTRODUCTION

Bichat (1802) described branches, variable in number and site of origin, from the femoral nerve to the femoral artery. These branches are commonly referred to in French literature as the 'nerve of Bichat'. Goering (1836), Cruveilhier (1852), Luschka (1863), Sappey (1871), Frey (1876), and Rudinger (1878) added further details concerning the topography of the vascular branches from the femoral nerve. A branch from the genito-femoral nerve was traced to the walls of the femoral artery by Luschka (1863), Sappey (1871), Krause (1879), Thane (1897), and Hovelacque (1927). Thane (1897) subdivided the vascular contribution of this nerve into two parts: from (*a*) the genital branch (N. spermaticus externus) a filament was supplied to the external iliac artery, and from (*b*) the femoral branch (N. lumbo-inguinalis) a contribution was provided to the femoral artery.

Nerve filaments derived directly from the lumbar plexus were traced to the femoral artery by Schwalbe (1881). Pacinian corpuscles were observed along the course of the femoral artery by Luschka (1863), and Krause (1879). Vascular nerve filaments were found arising from the *motor* branches of the femoral nerve, e.g., the nerve to rectus femoris, by [illegible]s (1914).

Recent investigators have emphasized the difficulty experienced when attempting these fine dissections in formalin-hardened material (Verglas, 1925; Hahn and Hunczek, 1925; and Lazarthes, 1939). More rigid control of the work was accepted, notably in the routine histological examination of the dissected nerve strands, by Hirsch (1925), Blair, Duff, and Bingham (1930), Coates (1931–2), and Woollard and Weddell (1934). Woollard (1926) produced the classic description of modern literature upon which many subsequent publications have been based. His conclusions were briefly as follows:—

a. There exists upon the abdominal aorta a rich plexus of nerve-fibres derived from the adjacent sympathetic chain ganglia and therefore of non-myelinated nature. This plexus is traceable along the iliac and femoral vessels whereupon the fibres gradually disappear to be replaced by small filaments derived from local peripheral nerve-trunks. These latter filaments are predominantly of myelinated nature in the ratio of 4 to 1.

b. Degeneration experiments showed the non-myelinated fibres to belong to the sympathetic system.

Woollard regarded these results as confirmatory of those by Lapinsky (1905), who had found the femoral nerve contributing but few vasomotor fibres to the femoral artery. Woollard found no evidence of ganglia or other neural mechanism surviving in the vessel walls after proximal denervation. He conducted these investigations upon laboratory animals, using methylene blue intravitam staining. It is perhaps unfortunate that later workers have, on occasion, appeared to regard these results as applicable to man, not only in principle, but also in detailed morphology.

Kerper (1927) performed experimental denervations in the dog, and concluded that the large myelinated fibres observed in the tunica adventitia were sensory, and were derived from the posterior roots of the spinal nerves. The small myelinated and non-myelinated fibres were derived from two sources, viz.: (*a*) the posterior nerve-roots, and (*b*) the ganglionated sympathetic chain. Kerper claimed that a combined posterior nerve-root section and unilateral sympathectomy rendered an artery devoid of nerve-fibres. These results appear never to have

been confirmed. They are of considerable interest because they indicate (*a*) a strictly unilateral innervation of hind-limb vessels on both the afferent and efferent sides, and (*b*) that the theory of short reflex arcs (as postulated by Leriche, 1928) persisting after operative denervation, is untenable.

Leriche and Fontaine (1928) urged further anatomical study, observing that knowledge of the vascular nerves had not been markedly advanced since the work of Potts (1914), and of Kramer and Todd (1914). They postulated the existence of intrinsic intramural centres with afferent and efferent components forming a true reflex arc independent of the extrinsic nerves. These last they regarded as forming long-chain association fibres enabling local vascular changes to be harmonized with the requirements of the general circulation.

Coates (1931) suggested that, as the first evidence of recovery following peripheral nerve injuries concerns the disappearance of trophic changes, this might be due to the regeneration of vascular nerves. Delmas and Laux (1931) considered the transition from a visceral to a parietal type of innervation took place on the external iliac artery. Stopford (1931) summarized current views regarding the anatomical arrangement of nerves and nerve-fibres supplying peripheral blood-vessels of the lower limb. Such arrangements comprised : (*a*) non-myelinated sympathetic vasoconstrictor fibres reaching the vessels via the axial arterial plexus, or from peripheral nerves, with a control centre situated in the vicinity of the tuber cinereum ; (*b*) myelinated fibres, exclusively from peripheral nerves, supplying the vessels of the more distal parts of the limb, and with cell stations situated in the posterior nerve-root ganglia. These myelinated fibres might be concerned with vasodilatation and with those phenomena usually attributed to the antidromic impulse. Stopford believed there was ample proof that these myelinated fibres provided a pathway for axon reflexes, and thought that some of these fibres might subserve deep sensibility. Sheehan (1932) observed that sympathectomy gives uncertain results in the upper limb. The explanation advanced by Lewis (1932) that this is due to pathological change in the peripheral vessels failed to account for the much better results obtainable in the lower limb.

Telford and Stopford (1932) deduced that cutaneous blood-vessels must have the same innervation as the associated skin itself because soon after a nerve lesion the resultant area of anæsthesia may be defined by the observable zone of hyperæmia. This phenomenon, however, gave no clue to the distribution of vasoconstrictor fibres to the deep vessels. Telford and Stopford suspected this distribution to be complex and variable (as indeed is deep sensibility) and to be non-segmental. Woollard and Phillips (1932) believed that branches from peripheral nerves to the blood-vessels of the upper limb were distributed in a segmental manner. They found no evidence of nerves to the blood-vessels of muscles, and it was inferred that these, if present, must be few in number. Theis (1933) performed the phenomenal task of 260 operations, mostly sympathectomies, upon 110 dogs. His results showed that the sympathetic control to this animal's hind limb was strictly unilateral.

Kuntz (1934), in a comprehensive review of the subject, quoted the work of Kerper (1927), and concluded that the total extrinsic nerve-supply to blood-vessels was far greater than at one time believed. Woollard and Weddell (1934) showed that greater constrictor tonus at the periphery of the limb could have an anatomical basis, but they regarded the dissection of nerve-fibres to blood-vessels as unreliable. They admitted, however, that their material was not fresh, often of advanced age, and that their lower limb material was from patients suffering from Buerger's disease.

Dolgo-Saburoff (1936) claimed to have discovered ganglion cells in the walls of arteries. The presence of such has been denied, and the suggestion made that these 'ganglion' cells are merely misidentified Rouget cells (Le Gros Clark, 1945). Leriche (1939) described vascular nerves joining an axial limb vessel at intervals along its length, more or less at right angles to its long axis. Woollard, Weddell, and Harpman (1940) showed nerve-fibres to be present in the adventitia of blood-vessels following sympathectomy in experimental animals : the surviving fibres were morphologically similar to fibres known elsewhere to transmit pain impulses. Le Gros Clark (1945) believed arteries to be variable in the degree of complexity of their innervation : muscular arteries for example had a far more feeble innervation than had cutaneous vessels. Nevertheless, experimental evidence showed that even muscular arteries did respond to sympathetic stimulation.

Millen (1948) studied the innervation of the smaller arteries in the ear and stomach of the rabbit, using methylene blue technique. A single nerve filament was found to supply a short length of small arteries, or a greater span of arterioles. Millen suggested that sensory receptors in the tunica adventitia of the larger blood-vessels might function as tension recorders, whilst the deeper plexus between the tunica adventitia and tunica media might serve vasodilatation and pain reception.

MATERIAL AND METHODS

Perusal of the literature indicates that certain of the vascular nerves are of such considerable dimensions as to be accessible for dissection by normal methods using adult material preserved in the routine manner. However, the numerous references to difficulties encountered when dissecting the finer ramifications of such nerves in adult limbs, and comments concerning the dense nature of the perivascular connective tissue in pathological material, led to the employment for the present investigation of fresh full-term fœtuses.

Eighteen lower limbs from full-term human fœtuses were used, together with the hind limbs of an adult female rhesus monkey (*Macacus mulatta*). The circulatory system of each fœtus was washed through with 25 per cent methylated spirit ; the pelvis was then split sagittally and the hind quarter preserved in 20 per cent spirit. This method of fixation minimizes the hardening of connective tissue and facilitates the dissection of fine nerve filaments.

Dissection under fluid with the aid of a binocular microscope was performed according to a modification of Mitchell's (1935) water-infiltration technique.

A magnification of × 5 diameters was employed for the initial isolation of the larger vascular nerves: Histological examination was employed throughout to confirm dissection findings (*see Fig.* 151 b).

FIG. 143.—Photograph of dissection to show nerve filaments forming a plexus upon the femoral artery and vein.

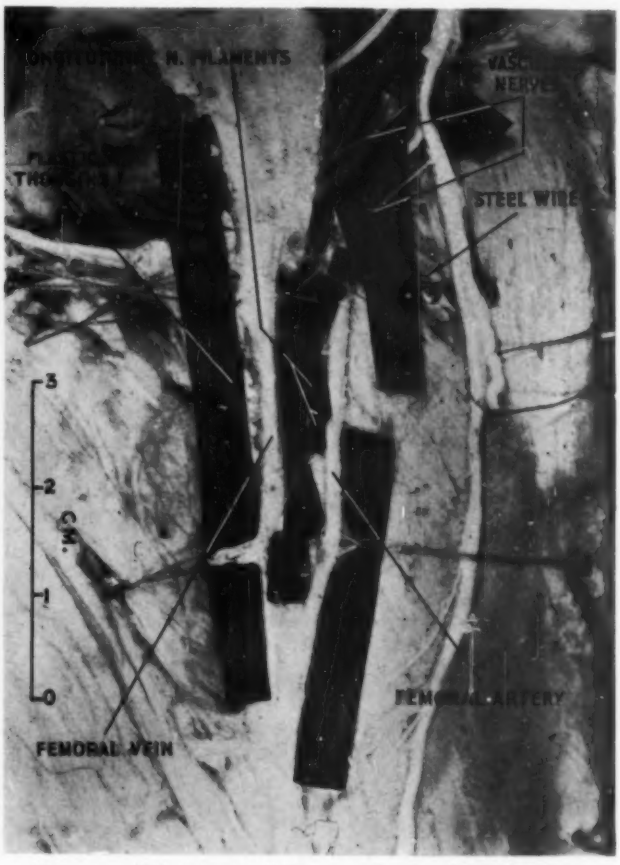

FIG. 144.—Enlargement of *Fig.* 143, showing femoral artery and vein retracted to display perivascular nerve plexus.

this was later increased to × 10 for the dissection of the finer terminal nerve filaments, whereupon arm-rests and instrument holders became essential to obviate hand tremor. It was found that the finer vascular nerves often remained almost invisible after isolation of their parent stems until some underlying contrast medium was introduced for their more emphatic display: black plastic thonging which may be obtained in $\frac{1}{4}$-in.-wide strips was found convenient for the purpose (*Figs.* 143, 144).

Retraction of the perivascular tissues was essential to the identification of these very tenuous and delicate vascular nerve filaments, and was achieved by the use of stay sutures, using the finest surgical silk introduced on a fine curved needle. As such sutures multiply rapidly as dissection proceeds, as their position requires frequent alteration, and as spun silk tends, on withdrawal, to catch and tear the most delicate nerve filaments, this material was replaced by $\frac{1}{1000}$-in. stainless steel wire (*Fig.* 144).

Experience soon taught that vascular nerve filaments once identified were rarely, if ever, damaged by subsequent manipulation. They were distinguished macroscopically from adjacent lymphatic vessels by their more sinuous course (*Fig.* 145), and their neural identity was established by their dissection back to the parent peripheral nerve-trunk.

The employment of full-term fœtuses preserved in the manner outlined enables the dissection of the

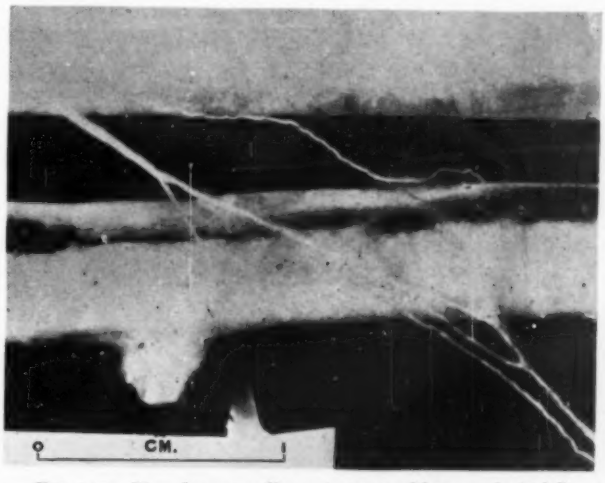

FIG. 145.—Vascular nerve filaments approaching a main (axial) artery as seen under the dissecting microscope.

vascular nerves to be performed with far greater accuracy than obtains when adult material is used. For it is possible to distend the delicate perivascular

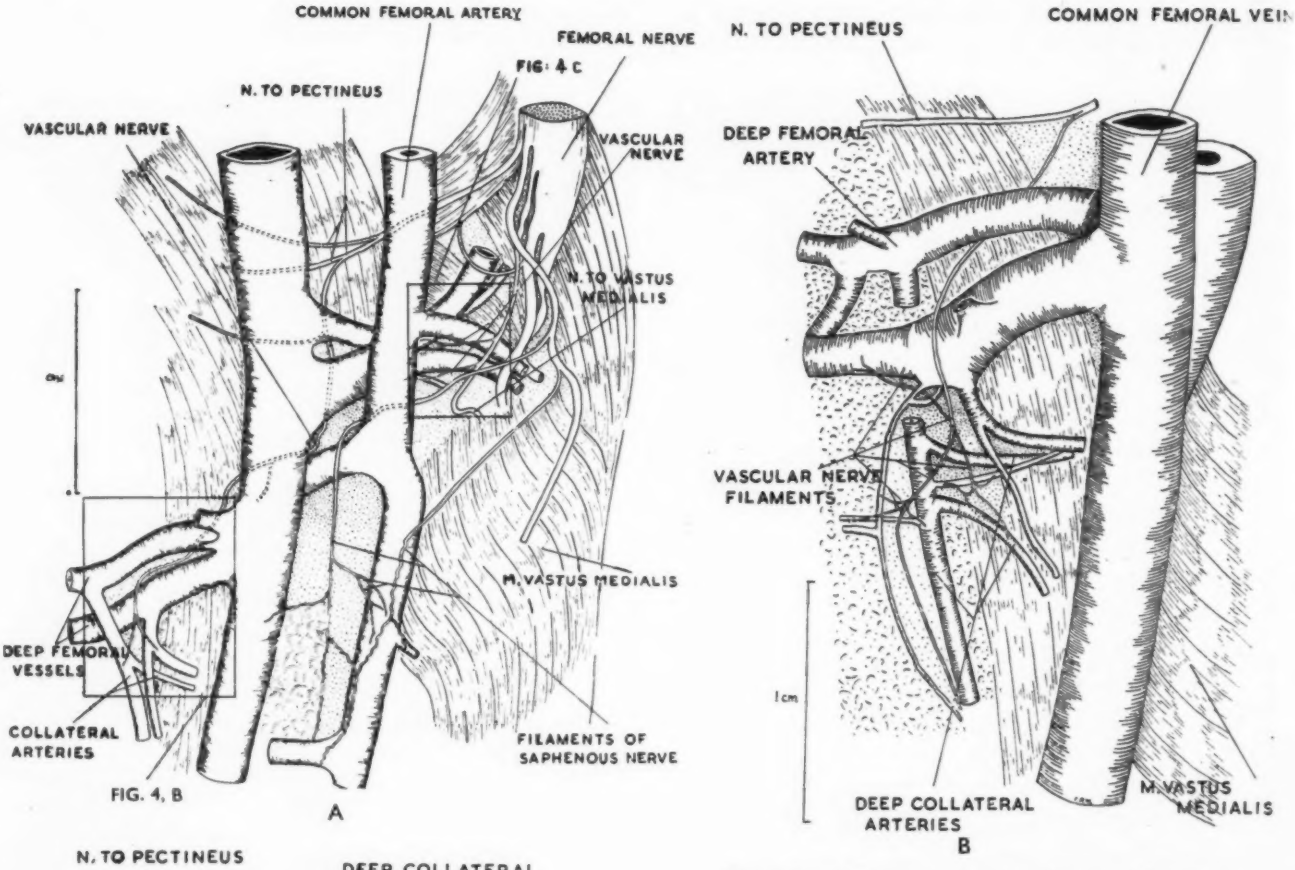

FIG. 146.—A, Diagram to show vascular nerve filaments to deep 'muscular' arteries (main axial vessel plexus not shown). B, Enlargement of inset in (A), showing arborizations upon deep 'muscular' arteries of a vascular branch from the nerve to M. pectineus. C, Enlargement of inset in (A), showing arborization upon lateral circumflex vessels of filaments from the nerve to M. vastus medialis.

connective tissue by fluid from a fine hypodermic needle and to trace nerve filaments into the tunica adventitia. Such a feature commands particular interest in the femoral region in view of the comments of many investigators concerning the barrier presented by the femoral sheath to such dissection in adult material.

FINDINGS

1. The Gross Morphology of the Vascular Nerves in the Femoral Region.—

a. Nerve to M. Pectineus.—The nerve to pectineus was found to give branches to the common femoral vessels in 15 of the 20 dissections; these short nerve filaments arose from the nerve to pectineus as this passed behind the femoral sheath. In 14 cases a long, slender nerve filament arose from the nerve to pectineus on the medial side of the femoral sheath. This delicate nerve branch ran distally across the M. pectineus to join the deep femoral and medial circumflex femoral vessels (*Fig.* 146 A). A fine twig from the nerve to pectineus formed arborizations upon the deep external pudendal artery in two specimens.

b. Intermediate Cutaneous Nerve.—The intermediate cutaneous nerve of the thigh contributed

branches to the common femoral artery in 10 dissections. These delicate filaments joined the perivascular nerve-plexus after a short, direct course.

c. The Medial Cutaneous Nerve.—The medial cutaneous nerve of the thigh provided branches to the common femoral vessels in 13 cases, and to the superficial femoral vessels in 15 of the dissections. Branches were identified in 4 cases to the deep external pudendal and medial circumflex femoral vessels.

d. The Long Saphenous Nerve.—Despite its close anatomical proximity to the main axial vessels, the long saphenous nerve was not observed to give branches to the common femoral artery or vein. A branch to the superficial femoral artery was found in 13 cases, and 2 delicate filaments ran to the lateral circumflex femoral vessels in 2 specimens (*Fig.* 147). In 14 of the 20 dissections the distal part of the superficial femoral vessels received multiple branches from the saphenous nerve (*Fig.* 148).

e. Deep Part of the Femoral Nerve.—The deep, muscular component of the femoral nerve provided vascular branches in 14 cases. These took the form of a comparatively large bundle which were distributed to the common femoral and deep femoral vessels. Those to supply the common femoral vessels took a recurrent course, passing distally around the bifurcation of the common femoral artery, whence they turned and followed the common femoral vessels as far as the inguinal ligament

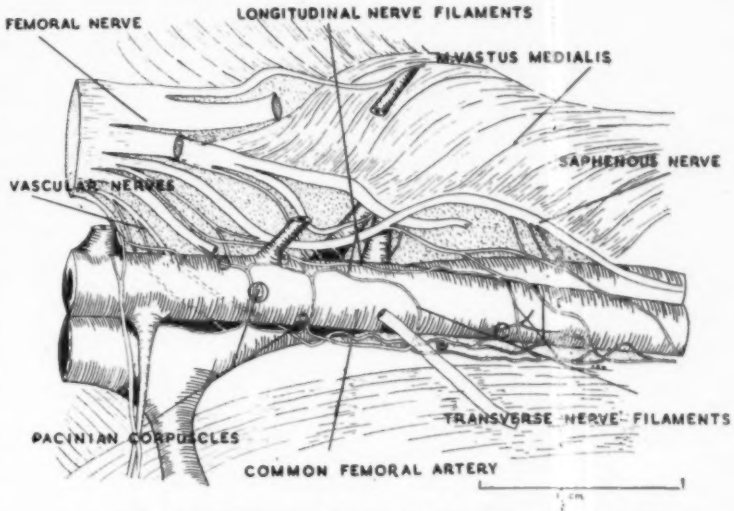

FIG. 147.—Diagram to illustrate the minute appearances of the neural plexus upon the femoral artery and vein.

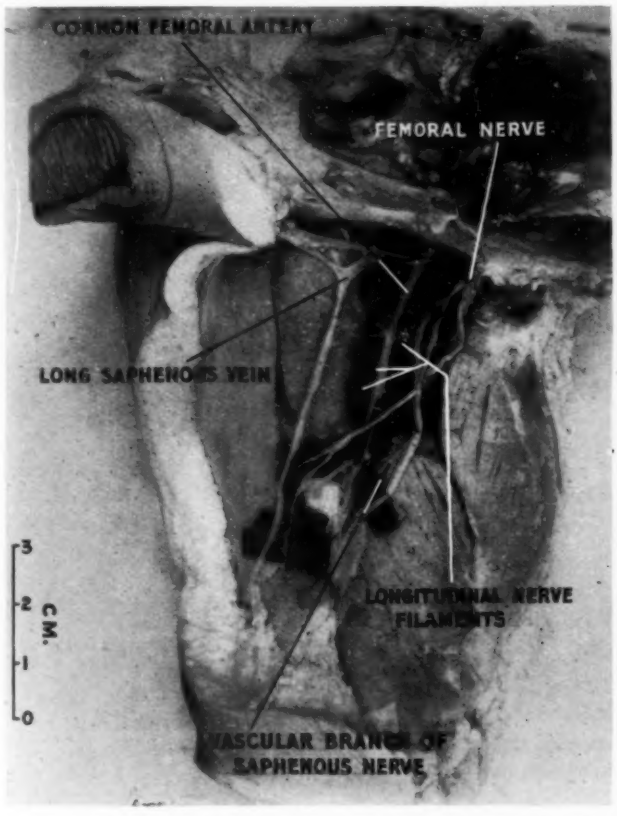

FIG. 148.—Photograph of dissection showing vascular branches recruited from the femoral and saphenous nerves. The longitudinal nerve filaments that accompany the femoral artery are evident.

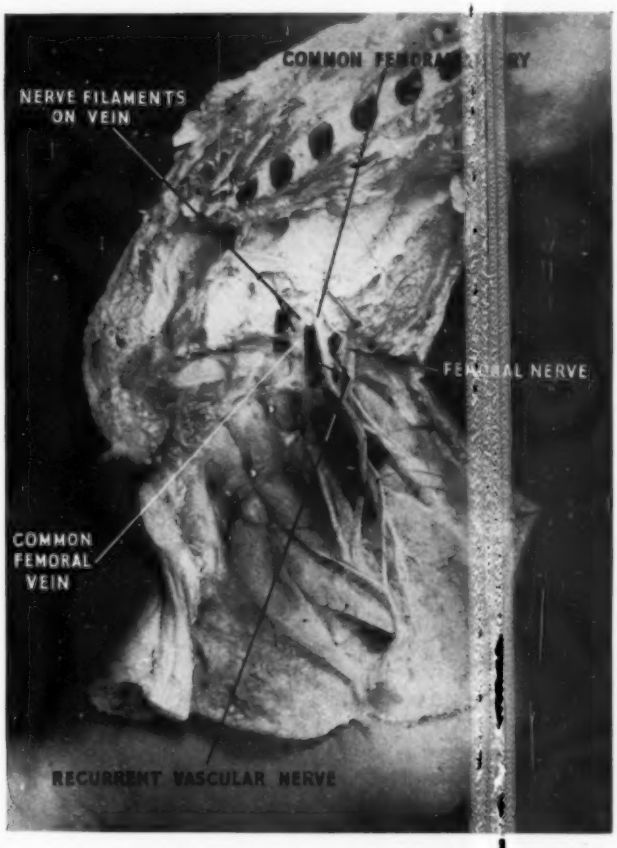

FIG. 149.—Photograph of dissection showing recurrent vascular nerve filaments with branches of distribution to the common femoral vein.

(*Figs.* 149, 150). In 10 dissections filaments were observed arising from the individual motor nerves,

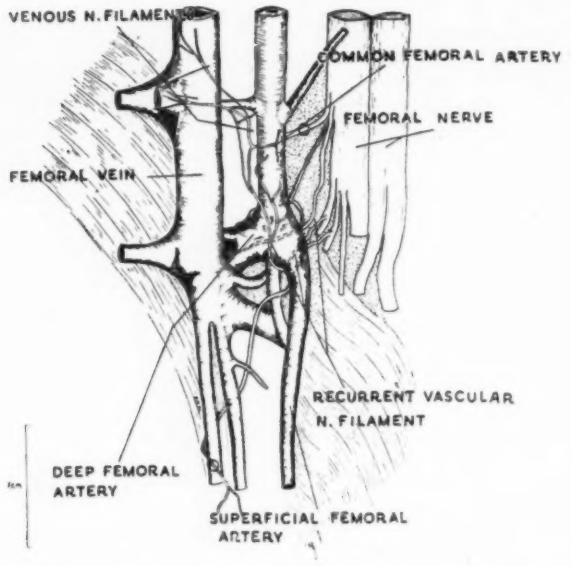

FIG. 150.—Diagram to illustrate the origin, course, and distribution of a recurrent vascular nerve.

e.g., nerve to M. vastus medialis, to supply the lateral circumflex group of vessels (*see Fig.* 146 A, C).

j. The Genito-femoral Nerve. — The femoral branch of the genito-femoral nerve gave a branch to the deep external pudendal artery in 4 cases. No evidence was obtained of any contribution from this nerve branch to the common femoral vessels, and it was inferred that the ' vascular ' fibres of the genito-femoral nerve are contained almost wholly within its genital branch for distribution, together with the ' para-iliac ' nerve (Wilde, 1949) to the external iliac artery. The enormous accession of vascular filaments derived from the femoral nerve, many of which are directed to the common femoral artery immediately distal to the inguinal ligament, is a most striking feature of every dissection. Direct continuity between the extramural plexuses upon the external iliac and common femoral vessels is, however, confined to one or two minute nerve filaments (Wilde, 1951). Thus the findings of Hirsch (1925) are fully substantiated in this respect.

2. The Minute Appearances of the Vascular Nerve Filaments in the Femoral Region.—

a. The principal vascular nerves to the common femoral and superficial femoral vessels form an adventitial plexus thereon (*Figs.* 144, 147, 148). This plexus is characterized by a number of exceedingly delicate nerve filaments disposed longitudinally upon the vessel walls, united at regular intervals by secondary loops (of even finer filaments) disposed transversely to the long axes of the vessels, the whole being reinforced at successive points by extraneous recruitment from local branches of the femoral nerve. In the human material used encapsulated nerve-endings were frequently seen upon the filaments of the adventitial plexus (*Fig.* 147). Higher magnification established that these end-organs were in structural continuity with the vascular nerve filaments; histological section showed them to be typical Pacinian corpuscles (*Fig.* 151 A). In the rhesus material studied no evidence of such encapsulated nerve-endings was obtained.

b. The numerous deep collateral and muscular branches derived from the deep femoral and circumflex femoral group of arteries are innervated in a different and quite characteristic fashion. Exceedingly tenuous nerve filaments originate from the motor branches of the femoral nerve, pursue a complex course, which (in the fœtus) may exceed 1 cm., and end as terminal arborizations upon the walls of the deep collateral arteries. *Fig.* 146 C shows nerve filaments arising from the nerve to M. vastus medialis

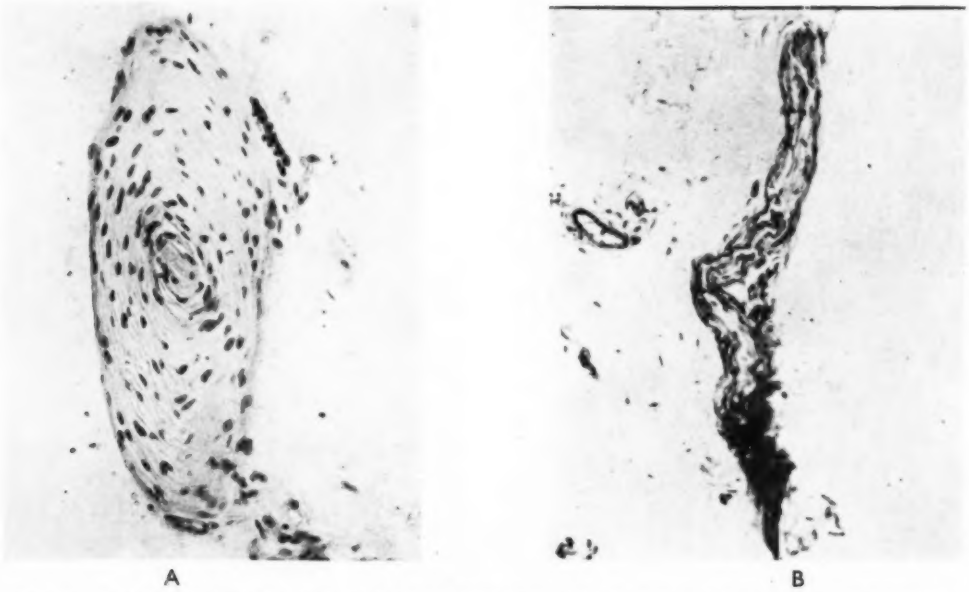

FIG. 151.—Photomicrographs: A, A Pacinian corpuscle (× 260); B, A vascular nerve filament (× 130).

and forming terminal arborizations upon the secondary branches of the lateral circumflex femoral artery.

The muscular branches of the medial circumflex femoral and deep femoral vessels also derive their vascular nerve filaments from motor branches of the femoral nerve. *Fig.* 146 B shows a vascular branch from the nerve to M. pectineus supplying the secondary and tertiary branches of the deep femoral artery. The figure depicts the complex course and surprising length of these very tenuous nerve filaments. There is no evidence of Pacinian corpuscles

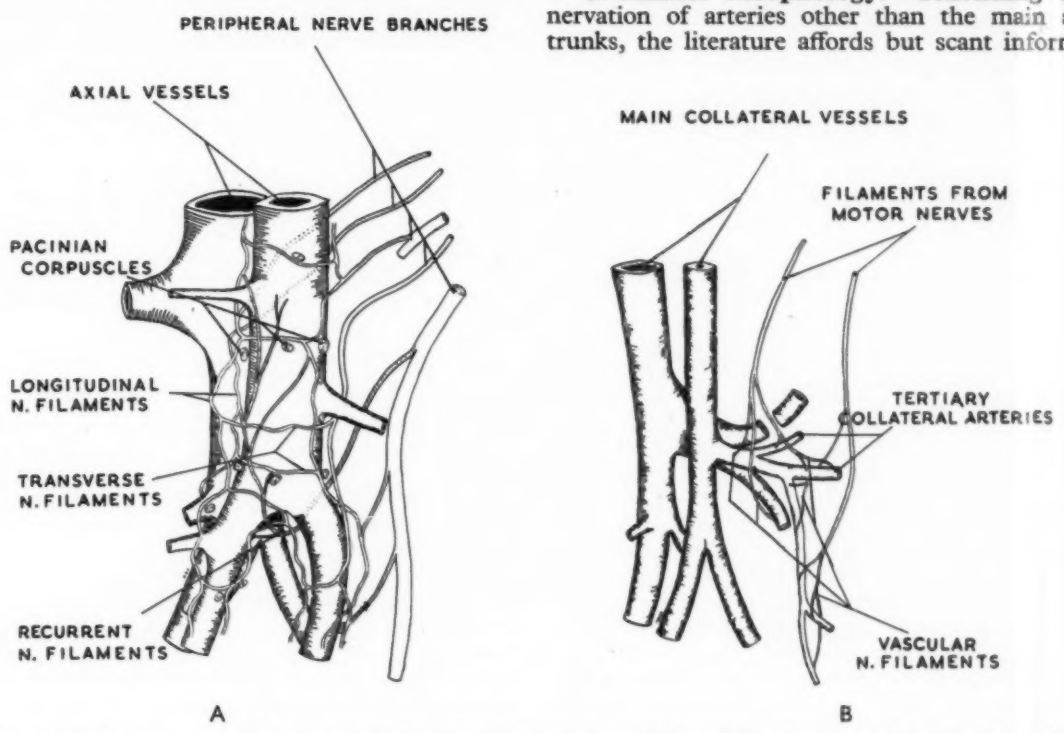

FIG. 152.—Diagram to contrast the perivascular neural patterns of: A, Main axial blood-vessels, and B, Deep 'muscular' and collateral arteries.

along the course of these vascular nerves, nor upon the walls of the deep muscular arteries. *Fig.* 152 is a diagram to illustrate the contrasting neural patterns of (*a*) the perivascular plexus upon main axial blood-vessels, and (*b*) the long and discrete nerve filaments with terminal arborizations upon the walls of deep muscular arteries.

DISCUSSION

1. Gross Morphology.—The larger, more robust vascular nerves, e.g., those from the genito-femoral nerve or from the main trunk of the femoral nerve, are accessible to any careful dissector using adult cadaveric material preserved in the routine manner. Their illustration, by previous observers, proves similar to the condition shown in *Fig.* 149 where a stout nerve-bundle is depicted penetrating the sheath of a vessel. Using the material and methods herein outlined, however, the substantial vascular nerves may be traced in continuity to the adventitial plexus upon main axial blood-vessels (*Figs.* 144, 148), and to the rich arborizations on the secondary and tertiary deep collateral arteries (*Fig.* 146).

The presence of vascular, or perivascular, pathological change constitutes a formidable, sometimes insuperable, obstacle to such dissection. Indeed, conditions such as diffuse obliterative arteritis may weld associated arteries, veins, and nerves into a solid continuum situate amidst a mass of dense, sclerotic fibrous tissue; and in this connexion it is not unremarkable that so many investigators should have failed in their attempted dissections of the vascular nerves in limbs amputated for Buerger's disease.

2. Minute Morphology.—Concerning the innervation of arteries other than the main arterial trunks, the literature affords but scant information, and such accounts consist mainly of general statements to the effect that the deep vessels manifest a meagre innervation of doubtful physiological significance. Present dissections, however, indicate the presence of a liberal innervation to those deep arteries supplying the skeletal muscle of the thigh, and fully support the experimental conclusions by Barcroft, Bonnar, Edholm, and Effron (1943), who obtained an increase of 87 per cent in the blood flowing through the deep forearm vessels after blocking the motor nerves in human subjects.

It is notable that the nerves supplying, and accompanying, these 'muscular' vessels are devoid of the encapsulated nerve-endings which constitute so obtrusive a feature of the adventitial plexus of the principal axial vessels.

3. The Relation of Structure to Function.—The demonstration of the presence of vascular nerves naturally leads to the positing of questions regarding their function: (*a*) Are such nerves predominantly motor or sensory? (*b*) Do they constitute a receptor for some, at least, of the elements of deep sensibility? (*c*) Do they provide information as to the neural control of arteries to skeletal muscle?

a. Are Such Nerves predominantly Motor or Sensory?—For many years the vascular nerves were, not unnaturally, considered to constitute an efferent pathway to blood-vessels from the sympathetic-chain ganglia. Much experimental evidence was accumulated to show the presence of vasoconstrictor fibres in the peripheral nerves, e.g., the vasodilatation that follows their paralysis. Electrical stimulation of the sympathetic chain or of the distal ends of cut peripheral nerves, however, gave somewhat conflicting results; such procedures might elicit vasoconstriction or contrariwise an 'active' vasodilatation. Hence the introduction by Bayliss (1900–01) of the theory of vasodilator nerves, a matter still *sub judice*. But the later suggestion that some of these fibres might be sensory in function, having their cyton situate in the posterior nerve-root ganglia, appeared more susceptible of proof since many of the fibres are myelinated. Kerper's (1927) experimental division of posterior nerve-roots appears to offer satisfactory proof that these myelinated fibres are indeed sensory. The presence of numerous encapsulated nerve-endings upon, and in neural continuity with, the filaments of the adventitial plexus strongly supports this inference.

b. Do They constitute a Receptor for Deep Sensibility?—It may be that the adventitial plexus of main axial blood-vessels forms a part of the anatomical apparatus subserving deep sensibility, for the associated presence of Pacinian corpuscles is a presumptive indication of ability to record deep-pressure stimuli. Although no positive information as to this can be expected from a topographical study of neural components, the hypothesis is strikingly supported by the observed absence of Pacinian corpuscles along the iliac vessels, where protection from deep pressure stimuli is afforded by the anterior abdominal wall. A series of 20 dissections to demonstrate the perivascular plexus of the common and external iliac vessels has shown (Wilde, 1951) the complete absence of Pacinian corpuscles in relation to the common iliac vessels, and the sudden appearance of such encapsulated nerve-endings at the termination of the external iliac vessels beneath the inguinal ligament.

c. Do They provide Information as to the Neural Control of Deep Arteries to Skeletal Muscle?—The demonstration of filaments from motor nerves to the deep muscular and collateral arteries provides a structural basis for the experimental observations by Barcroft et al. (1943). These investigators concluded, from experiments whereby vasodilatation occurred following interruption of the vasoconstrictor fibres in motor nerves, that deep muscular arteries were to some extent controlled by the autonomic system. It might be supposed that intermittent claudication would be relieved by sympathectomy, or by the more selective division of motor nerves, e.g., nerve to M. pectineus, or nerve to M. vastus medialis (in the thigh). Clinical evidence indeed favours this hypothesis, Paterson Ross (1947), despite the undoubted influence of metabolites upon the development of hyperæmia in exercised muscle.

SUMMARY

1. The vascular nerves to the deep lower limb vessels may be rendered available for direct study by the application of a dissection technique, certain precautions being observed regarding the type of material used and the method of its fixation.

2. Human fœtuses and the rhesus monkey show a number of femoral vascular nerves derived from the femoral nerve-trunk and its immediate branches.

3. These vascular nerves supply the whole of the common femoral and the superficial femoral vessels from the inguinal ligament to the hiatus in M. adductor magnus. The femoral nerve may well constitute the exclusive source of innervation of these vessels, though no such formal conclusion is advanced on the basis of the present limited series of dissections.

4. A coarse nerve-plexus formed upon the main arterial and venous trunks is composed of longitudinal nerve strands intercommunicating at intervals by means of fine transverse filaments lying at right angles to the long axis of the vessels. This reticular formation is reinforced periodically by direct contributions from the principal local nerve-trunks.

5. In the human subject this adventitial plexus clothing the main axial femoral vessels is richly beset with Pacinian corpuscles. It is suggested therefore that functionally the plexus is largely sensory in nature and that it may constitute a component of the deep sensory pathways.

6. Vascular (i.e., perivascular) nerves are traceable from the main local nerve-trunks (or their branches to muscles) to the secondary and tertiary branches of the deep (and predominantly 'muscular') vessels such as the medial and lateral circumflex femorals. These nerves to 'muscular' arteries are devoid of Pacinian corpuscles. They afford an anatomical basis for the neural control of deep 'muscular' arteries.

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INTESTINAL OBSTRUCTION PRODUCED BY FAILURE OF INTESTINAL ROTATION

DESCRIPTION OF A CASE

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To understand the embryology leading up to the final anatomical disposition of the midgut, the reader is referred to the papers by Mall (1898), Frazer and Robbins (1915), and Dott (1923). Very briefly recapitulated, the process has been artificially divided for descriptive purposes into three phases. For embryological purposes, the midgut is the portion of the alimentary tract extending from the duodenal ampulla to the middle of the transverse colon. It is at first suspended in the midline of the embryo in its entire length from the posterior abdominal wall by a dorsal mesentery which contains and conducts the main blood-vessel and its branches, viz., the superior mesenteric artery. This vessel crosses the primitive gut at the point of emergence of the vitello-intestinal canal (which may remain as a Meckel's diverticulum in the fully developed fœtus). The gut head-ward of this point is referred to as pre-arterial, and caudal to it as post-arterial. The cæcum develops a little distal to the point of crossing. The line of the artery itself is the axis about which rotation occurs during the various developmental stages.

During the first weeks of development the capacity of the abdominal cavity is insufficient to accommodate the disproportionately growing liver, in addition to its other contents. Physiological herniation occurs at the umbilicus and the midgut is eventrated. At first the afferent or pre-arterial limb lies towards the head and the efferent limb is caudal as they enter and leave, respectively, the neck of the hernia. The first phase of rotation occurs at about the fourth week, when the growing mass of the liver displaces the upper abdominal contents. Consequently, the afferent or pre-arterial limb takes up a position to the right of the efferent one at the hernial orifice. Instead of lying in a truly sagittal midline plane, the dorsal mesentery has rotated in an anti-clockwise direction through 90°.

The second stage takes place rapidly about the tenth week. The midgut is withdrawn from the hernia. During the process of re-entry into the abdominal cavity, the pre-arterial gut takes precedence (it is thought that this is due to the relative bulk of the cæcum holding back the post-arterial loop). In entering the abdomen, the pre-arterial coils of gut are directed downwards (supposedly by the mass of the liver) behind the main artery. Further rotation of the dorsal mesentery occurs through 180°, making the total rotation 270°. The cæcum and terminal ileum, which re-enter last, thus come to lie in the subhepatic region.

The third and last stage is the attainment of the final position of the cæcum in the right iliac fossa, and the loss of the common mesentery of the midgut. The cæcum and ascending colon become fixed to the posterior abdominal wall. The duodenum also loses its posterior mesentery. Between duodenum and cæcum the dorsal mesentery acquires a secondary connexion to the posterior abdominal wall.

Failure or alteration of this sequence of events leads to abnormal disposition of the fully developed alimentary tract. According to Dott (1923), when the opening of the physiological hernia is abnormally wide, mass reduction of the hernia occurs. The whole midgut returns to the abdomen *en masse* and non-rotation results. The whole of the jejunum, ileum, cæcum, and ascending and transverse colon have a common mesentery. The cæcum is situated

in the upper abdomen. The disposition of the gut is as if development had ended at the second stage, say about the eighth week.

Should the opening be abnormally narrow, 'reversed' rotation occurs. This is because the post-arterial loop of gut re-enters the abdomen first and passes behind the main vessel. In the completed anatomy, the transverse colon is seen to be posterior to the duodenum. Examples of reversed rotation are described by King (1937), and by McIntosh and Donovan (*Case* 19) (1939).

Malrotation is less easy to understand. Cases are described of herniation of the pre-arterial segment of gut into the mesentery of the post-arterial portion. The mesentery of the post-arterial portion envelops the pre-arterial gut like a sac. Examples are described by King (1937), and by McIntosh and Donovan (*Case* 18) (1939). The pre-arterial gut has passed in front of the artery, but the post-arterial gut has made a normal attempt at rotation in these cases. Another variety of malrotation is described in which, due apparently to mass entry and non-rotation, the small intestine remains on the right side of the abdomen while the cæcum and ascending colon, having rotated normally, retain a mesentery and remain on the left side of the intestines. *Cases* 11 and 12 of McIntosh and Donovan (1939) are of this type of malrotation.

The conditions of malrotation, and particularly reversed rotation, must not be confused with 'situs inversus', a rare condition in which the anatomy is reproduced as a mirror image of the normal, along with dextrocardia and other reversals. McIntosh and Donovan report an example associated with a volvulus (*Case* 17).

The unusual position of the viscera would have little significance (except in the case of reversed rotation) were it not for two complicating features: First, the presence of a common mesentery suspending the whole midgut (from the duodenum to the transverse colon) by a narrow attachment to the posterior abdominal wall predisposes the gut to the mechanism of twisting; Secondly, in addition to failing to obtain the normal fixation to the posterior abdominal wall, persistent peritoneal bands and sheets are found. The commonest of these are in the region of the duodenum. Abnormal fixation of the second and third parts of the duodenum may be found. Also, abnormal attachments of the malplaced cæcum to the posterior abdominal wall may cross and constrict the duodenum. Numerous observers (Wakefield and Mayo, 1936; Waugh, 1928; Morton and Jones, 1936; Miller and Gage, 1930; Ladd and Gross, 1941; Woolsey, 1927; and others) have reported these adventitious adhesions. Clausen (1938) designates the condition 'hyperfixation of the duodenum' and stresses that the bands are congenital and not inflammatory.

The following is an account of a patient admitted with so-called 'volvulus neonatorum'.

CASE REPORT

A. R., female, aged one year ten months, was admitted to hospital on May 8, 1949.

PERSONAL HISTORY.—The patient was second in a family of four children. Her birth was normal at full term and her birth weight was 6 lb. 14 oz. During the pregnancy her mother's health was good.

The patient was breast-fed until she was five months old, after which she was given Full Cream National Dried Milk. She is stated to have cried excessively on account of 'wind'. For this her mother administered Nurse Harvey's 'Wind Mixture' and 'Cinder Tea' (Cinder Tea is prepared by immersing red hot cinders into a cup of cold water. After sieving, the water is ready for drinking by the patient). Her mother seems to think these measures were successful in 'getting up the wind'. She never vomited and at first weight was gained at the rate of one pound per week.

Previous Illnesses.—She had chicken-pox in 1947, measles early in 1948, and pneumonia in December, 1948. She had some abdominal pain during her illness in December, 1948. Her mother stated that she complained of occasional attacks of abdominal pain during the twelve months prior to this present illness. Apart from this, she always appeared to be a normal infant. Her mother says that she was never lacking in energy and played all the day. She slept in the knee-elbow position.

FAMILY HISTORY.—

Parents: At the time of the patient's admission the mother was 31 years of age. She had been married about ten years when the patient was born. Her health had always been good. During the previous twenty years she had been troubled with dyspepsia. A radiograph taken in 1946 or 1947 failed to demonstrate peptic ulceration. The only finding was ptosis of the stomach.

The father had always enjoyed good health.

Other Children:

1. An older sister, born Oct. 27, 1940, suffered from attacks of abdominal pain every few months, associated with mild tonsillitis. This girl has since had a laparotomy on account of her abdominal pain. There were enlarged discrete mesenteric glands. There was no evidence of malrotation of the gut. The final diagnosis was non-specific mesenteric adenitis.

2. A younger sister, born April 26, 1948—no serious illnesses.

3. A brother born Oct. 5, 1949. He is normal and healthy. So far he has been free from illnesses.

The patient's maternal grandmother was married twice. By her first husband she had several children—amongst them the patient's own mother. By her second husband there were fourteen children. One male child was born with " stoppage of the bowels " and died when only a few days old.

HISTORY OF ILLNESS.—The symptoms began suddenly during the morning of Wednesday, May 4. Her bowels acted normally on the morning of the onset of symptoms. The stool was light in colour and well formed. She had been taken out with her mother to the shops and on her return she went out to play in the garden. She was seized with a sudden attack of abdominal pain, vomited greenish material, and threw herself face downwards on to the ground. She came into the house and doubled herself up over the arm of the settee. Vomiting occurred again five or six times and copiously. She seemed fairly comfortable during the rest of the day and moved about the house. Periodically she lay on the settee. In the evening she ate a substantial meal of steak, onions, gravy, and potatoes. She slept with her sister and vomited once during the succeeding night. On the morning of May 5, she still seemed to have attacks of abdominal pain, and doubled herself up over the arm of the settee crying out for her mother at the same time. She vomited frequently all through the day. Her day was spent resting on the settee. She was given drinks only by mouth.

Again during that night she vomited frequently. She slept with her parents.

All Friday, May 6, was spent resting on the settee. She was given drinks, M. & B. 693, and milk of magnesia prescribed by her own doctor.

Saturday, May 7, and Saturday night, were passed as the preceding days—on the settee during the day and in

her parent's bed at night. She had to lean over the side of the bed frequently to be sick.

She had no urinary symptoms, no headaches, and no cough or respiratory distress.

She was admitted to Alder Hey Children's Hospital on May 8, having lost about 1 st. in weight, and there had been no bowel action since the onset of her illness.

ON ADMISSION.—She was a quiet and co-operative patient. Her temperature was 99·6° F., and her pulse-rate 126; the respiration rate was normal. There were a few scattered petechial spots on the skin over the front of her chest and abdomen.

Abdominal examination: There was no distension. Movements were not restricted and there was no visible peristalsis. On palpation, there was no muscle guarding, but she was slightly tender on deep pressure over the epigastrium. There was no palpable tumour present. There was no evidence of hepatic enlargement and the spleen could not be felt.

The heart, respiratory system, ears, nose, and throat, and the central nervous system were all normal.

No immediate diagnosis was made—and she was kept in for further investigation.

On May 9 her condition was unchanged. Her eyes were sunken but her lips and tongue were moist. The skin of the abdominal wall was markedly lax. There was slight cyanosis of the lips and cheeks and circumoral pallor. General lethargy and muscular hypotonus were apparent.

She had a soft, scaphoid abdomen and, apart from slight generalized tenderness, there were no helpful physical signs.

The cardiovascular system was normal except for regular extrasystoles once every three beats. Again, the nasopharynx and ears were normal. She had slight neck rigidity.

A small enema saponis was administered, and a few small fæcal masses were returned in the fluid.

She vomited again in the afternoon. Gastric lavage with normal saline solution was performed, and clear bile-stained fluid was recovered from the stomach. After this, she managed to keep down a few ounces of milk.

Her temperature was never higher than 91·5° F. during the day and her pulse-rate was between 100 and 120 per minute. The respirations were normal. Her blood-pressure was 104/50 mm. Hg.

On May 10 the clinical picture was unchanged. It was thought that her conjunctivæ were slightly icteric. Lethargy and muscular hypotonus were still evident. She seemed to have attacks of abdominal colic, but again there were no physical signs on abdominal examination. She was taking limited fluids by mouth and she did not vomit. There was clinical evidence (sunken eyes and lax skin) of considerable dehydration. She seemed to be emotionally unstable.

Laboratory investigations showed a white-cell count of 12,700 per c.mm. The differential count was normal. The erythrocytes appeared normal. The urine had a sp. gr. of 1006, *p*H 6·0. Albumin was present, but no abnormal renal deposits were seen.

On May 11 she again vomited copiously in the morning. There was no change in her general condition and she was put on a régime of 3-oz. glucose drinks hourly.

The blood chemistry confirmed the severe loss of sodium chloride from vomiting. The blood-urea was raised (108 mg. per cent), but there was no appreciable change in the alkali reserve. There was no disturbance of potassium metabolism.

Tuberculous meningitis, infective hepatitis, so-called 'cyclical vomiting', uræmia were each considered as a possible diagnosis before a surgical opinion was requested.

She was transferred to a surgical ward on the morning of May 11. She had had no bowel action for eight days. She had vomited frequently and copiously throughout and had suffered from repeated attacks of abdominal colic.

She was dull, apathetic, and wasted. Her eyes were sunken, her skin lax, and her features presented a pinched appearance. Her abdomen showed no distension, no visible peristalsis, and no limitation of respiratory excursion. Palpation gave no help in making a diagnosis and the rectum was empty.

The history of vomiting, the clinical picture and the changes in blood chemistry indicated a severe state of salt and water loss.

A provisional diagnosis of duodenal or high intestinal obstruction was made.

The following investigations were carried out:—(1) A straight radiograph of abdomen. (2) The two enema test. Two soap and water enemata were administered with a half-hour interval between them. (3) Barium meal and 'follow through' screening. (4) Barium enema.

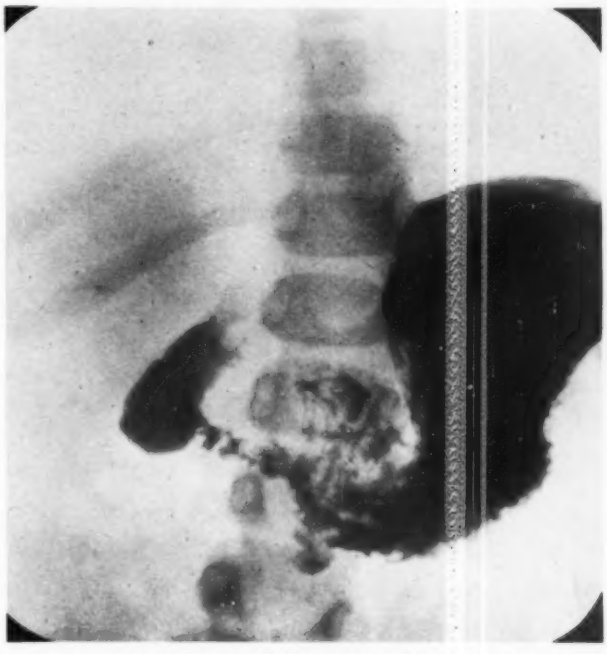

FIG. 153.—Radiograph of abdomen 90 minutes after barium meal. No barium has passed beyond the duodenum.

RESULTS OF INVESTIGATIONS.—

The straight radiograph showed no abnormal gas shadows or fluid levels which would have pointed to a diagnosis of intestinal obstruction.

Both enemata were returned without fæcal content. No flatus was passed with the second enema.

The barium meal resulted in important observations: Under the X-ray screen, the barium passed down the œsophagus without hindrance. The stomach outlined normally and barium entered the duodenum within a few minutes. At the end of 1½ hours no barium had passed the duodenum into the jejunum (*Fig.* 153).

The barium enema filled the rectum, the ascending colon, and the transverse colon. None would pass beyond the region of the right hypochondrium. There was no regurgitation into the lowest coils of ileum (*Fig.* 154).

It was evident that there was a complete obstruction in the region of the duodenum and a diagnosis of volvulus of the small intestine was made.

An intravenous drip of N/5 saline and 5 per cent dextrose was started and this was kept running throughout the subsequent proceedings until she was able to take fluids normally by mouth.

AT OPERATION.—This was performed on May 11. The anæsthetic was ethyl chloride followed by ether dripped on to an open mask.

The abdomen was opened by a right upper paramedian incision. A small amount of straw-coloured fluid was present in the peritoneal cavity. The stomach and duodenum were both markedly distended. The distension extended as far as the terminal portion of the third part of the duodenum. Near the duodenojejunal flexure the mesentery of the small intestine lay stretched across the

FIG. 154.—Radiograph of abdomen after barium enema. Colon filled as far as right hypochondrium. No barium beyond this point.

duodenum, compressing it against the structures on the posterior abdominal wall.

The small intestines were brought out through the incision on to the surface. There was found to be present a torsion of the mesentery of the small intestine, cæcum, and ascending colon. The coils of intestine were moderately distended, but only slightly altered in colour. Although venous congestion was present, there was no evidence of loss of viability. By rotating the mesentery two and a half turns in an anti-clockwise direction, the volvulus was untwisted. When the mesentery was completely untwisted, the cæcum lay naturally on the left side, about the level of the umbilicus. The mesentery then ceased to constrict the duodenum (*Figs.* 155, 156).

The untwisting of the mesentery, and the release of the third part of the duodenum, however, failed to relieve the intestinal obstruction. The first 9 in. of jejunum were bound down to the posterior abdominal wall by a vascularized sheet of peritoneum. The sheet not only fixed this portion of jejunum, but also constricted it. The jejunum was liberated by division of the sheet between a series of artery forceps parallel with the gut and immediately to the right of it. This portion of the jejunum was seen to fill after division of the constricting sheet.

It was seen that the ileum, jejunum, cæcum, and colon as far as the sigmoid were suspended from the posterior abdominal wall by a common mesentery whose attachment to the posterior abdominal wall was very limited in extent. The entire length of posterior wall attachment extended from the lower margin of the neck of the pancreas to a point about 9 in. below the duodenojejunal junction. The first 9 in. of jejunum were fixed to the posterior abdominal wall (*Fig.* 156).

The vessels traversing the sheet of peritoneum fixing the upper jejunum to the posterior abdominal wall were numerous, but fine, and were not associated with the blood-supply to the alimentary tract. Their division in no way endangered the gut.

The abdomen was closed in layers in the usual way.

PROGRESS.—Following the operation, her general condition was very precarious. There were frequent extrasystoles and her pulse-rate was between 130 and 140 per minute. Her condition showed little change on the first, second, and third post-operative days. During the afternoon of May 15 (fourth day) she vomited a large amount of greenish fluid. There was marked abdominal distension and still no bowel action. An enema saponis was given and some fæces and flatus were passed. A second enema half an hour later was returned clear and without flatus. A barium meal observed under the screen demonstrated that there was still a complete hold-up in the stomach and duodenum.

SECOND OPERATION.—This was performed on May 15. The abdomen was re-opened through the first incision. Free fluid was again present. The stomach and duodenum were again seen to be distended. It was discovered that the sheet of peritoneum described at the first operation binding the first part of the jejunum to the posterior abdominal wall had not been divided in its lower two inches. There was therefore still a mechanical obstruction present which was relieved only on division of the sheet. There was no evidence of recurrences of the previous volvulus. The abdomen was carefully resutured.

PROGRESS.—She vomited once on the morning of May 16 but her general condition gradually improved. The abdominal distension diminished. Borborygmi were audible and her first normal spontaneous bowel action took place on May 19.

Penicillin 500,000 units per day was given for six days and sulphamethazine 0·75 g., and subsequently 0·5 g. four-hourly for four days from May 16.

Adequate fluid replacement was maintained throughout from May 11 until May 19 by means of N/5 saline and 5 per cent dextrose. One pint of Hartmann's solution was also given. Continuous gastric suction was performed following the second operation and maintained for three days.

From May 19 her progress was satisfactory. The stools were at first loose—but later bowel action occurred normally once or twice daily. The wound healed and she was discharged from hospital on June 8.

She was seen last in June, 1950, and was quite well and had had no further symptoms.

Figs. 155, 156 demonstrate the findings at operation and the disposition of the viscera after unwinding the volvulus.

DISCUSSION

Very few observers have had the opportunity of seeing more than one or two of these cases.

Dott (1923) reviewed 48 cases in 1923, 5 of which were personal cases. He pointed out that symptoms had occurred in only 13 of these. It is to be inferred that not all cases having one of these anomalies presents with symptoms. A person may go through life with the condition unrecognized or, if found, it is incidental.

The most exhaustive study of the subject from the clinical aspect was made by Gardner and Hart

(1934), who reviewed 103 cases collected from the literature and added 2 of their own. They found that 55 or 52 per cent of these were newborn when first seen or had had symptoms since their earliest days after birth. Gardner and Hart found that of the 105 cases reviewed, 88 or 84 per cent were complicated by the occurrence of volvulus. In only 23 of the cases was the whole of the midgut (e.g., jejunum to mid-transverse colon) involved in the twisting of the common mesentery. They found that 17 cases were in the age group 3–27 when they first presented with symptoms.

FIG. 155.—Condition found at operation. Mesentery twisted through two and a half turns in clockwise direction. Duodenum constricted.

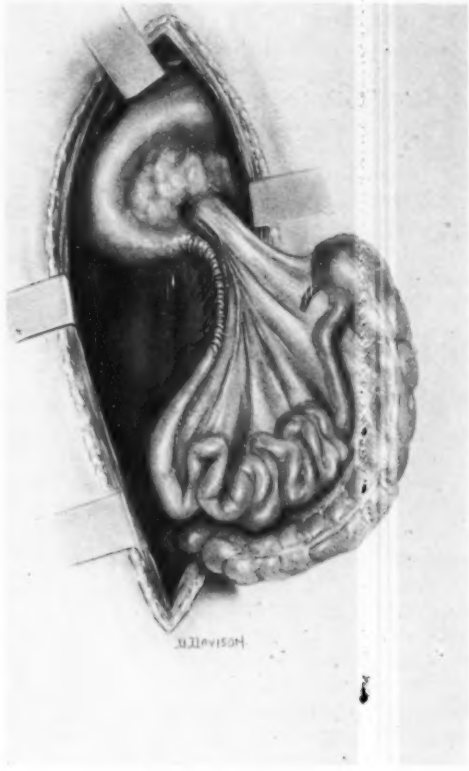

FIG. 156.—Condition after untwisting volvulus. First part of jejunum still constricted by congenital peritoneal sheet.

McIntosh and Donovan in 1939 described 20 personal cases, 18 of which were visualized at operation: 2 improved without operative intervention. Their cases presented examples of every known type of abnormality. Thirteen of the cases were complicated by volvulus; 85 per cent of the cases were seen in the first two weeks after birth. One interesting case described by them was a 14-day-old girl with duodenal obstruction and volvulus of the common mesentery. The volvulus was untwisted and the patient made a good recovery. The condition recurred at one and a half months and required a second exploration. She had a recurrence of symptoms at five years, but operation was avoided. At the time the account was written, she was 14 years of age and free from symptoms.

The possibility of recurrence of symptoms makes it necessary to consider the advisability of performing some fixation of the movable gut in order to prevent at least the recurrence of volvulus. This point will be considered again later.

Jones and Morton (1938), postulate a view that in some of the cases there are periodic attacks superimposed on a background of ' marginal disturbance '. The marginal disturbance is partial duodenal obstruction and the superimposed attacks are caused by the occurrence of volvulus. They consider that the patient may recover spontaneously; when he vomits he automatically decompresses the upper alimentary tract.

Marx in 1939 described 2 cases: one, a boy of 16 years who had suffered three years from attacks of acute abdominal pain precipitated by long periods of standing. In the later attacks vomiting occurred. Duodenal obstruction was demonstrated by radiography. No operation was performed.

A case of volvulus occurring in a boy of 8 years is described by Pearse (1939), and operated on after his first attack of abdominal pain and vomiting. No attempt was made after reduction to fix the cæcum and no abnormal bands were discovered. The author hoped that there would be sufficient aseptic inflammatory reaction to cause spontaneous adhesions and prevent recurrence.

The early onset of symptoms is emphasized by Ladd (1941), who found that in a series of 44 cases, 26 were seen within the first three weeks of life.

The mortality statistics are interesting. Of the 105 cases investigated by Gardner and Hart (1934), 36 died without operation. Sixty-nine were subjected

to operation and 36 of these recovered. Of their 88 cases which had a volvulus, 32 died without operation. Fifty-six cases with volvulus were subjected to operation and 30 (53 per cent) survived and were relieved. In the 32 patients with volvulus neonatorum who presented within the first one month of life, 16 died without operation. Eight of the remaining 16 died after operation. Of the 17 cases presenting between the ages of 3 and 27 years, 8 died without operation during an exacerbation; of the 9 operated on, 8 (88 per cent) were relieved.

In McIntosh and Donovan's (1939) 20 personal cases, out of 16 operated on 6 died. The mortalities were due to delay, shock, inadequate relief, and post-operative infection caused by a leak from perforated gut.

Ladd is quoted by Arnheim and Felshin (1943) to have recorded a mortality of 10 out of 47 cases (21·6 per cent).

Generally speaking, the mortality-rate is high in these cases. This is caused partly by failure to operate: in cases which are operated on the mortality is about 50 per cent. The figures indicate the advisability of early exploration of all cases in which the condition is suspected.

Whatever the particular type of anatomical anomaly, the patients may be classified into three groups: (*a*) The true volvulus neonatorum presenting in the earliest days of life; (*b*) The intermediate and less acute patients subject to exacerbations; and (*c*) Patients in which the condition may be unrecognized or discovered accidentally.

The condition is rare and diagnosis is not easy. Two factors influence the clinical picture: First, the partial obstruction of the upper alimentary canal at the duodenojejunal flexure by the twisted mesentery crossing the duodenum, or by an adventitial peritoneal band or adhesion; and, secondly, the volvulus itself, which consists of a large mass of intestines the blood-supply of which is very precarious. Clinically, it is the duodenal obstruction which predominates in the early stages of the clinical course.

McIntosh and Donovan (1939) state that a pyloric mass may be palpated. This is the distended pylorus. The true volvulus neonatorum of very early life has to be differentiated from congenital hypertrophic pyloric stenosis, congenital atresia, intussusception, and obstruction caused by tumours or bands. In congenital pyloric stenosis vomiting is projectile, occurs immediately after feeding, and there is no bile in the vomitus. The other conditions usually have some characteristic clinical feature to distinguish them.

The older patients are often undernourished and underdeveloped. They give a history of recurrent attacks of pain and vomiting. These cases may be difficult to pick out of the numerous patients suffering from recurrent attacks of abdominal pain caused by other conditions, e.g., non-specific mesenteric adenitis. This applies particularly in children.

The diagnosis is usually settled by radiography. The use of barium is contra-indicated in infancy. The barium may run into the trachea, or a partial intestinal obstruction may become complete. One interprets the distribution of gas shadows and possible fluid levels shown in a straight radiograph. With older children, barium may be used as a meal or by enema and a hold-up can be demonstrated.

In the patient described, suspicion of the condition was aroused by the hold-up of barium in the duodenum and the failure of barium run in per rectum to fill the cæcum. Vomiting was the most persistent feature and constipation had become absolute. There was no evidence of abdominal distension or of visible peristalsis. It may be of some significance that during twelve months prior to the final illness, she had suffered from attacks of abdominal pain.

The findings at operation depend on the type of anomaly present. A good exposure is required either by means of a paramedian or a midline incision. The distended stomach and duodenum are usually the first most obvious finding. The cause of the obstruction is not obvious. It is necessary to get a clear and unobstructed view of the duodenum. To do this it is necessary to bring the whole of the gut on to the surface. This at the same time demonstrates the occurrence of a volvulus. The twisted gut is not usually distended, indicating that there is patency of the lumen at its distal end. The colour of the gut is surprisingly good, indicating that in spite of the number of rotations which can occur, there is not gross interference with the blood-supply. In regard to the direction and number of turns, these can occur in either a clockwise or an anti-clockwise direction. According to Gardner and Hart (1934), of 79 cases in which the direction was adequately described, in 70 per cent it was clockwise. The number of turns varies: four complete twists have been noted (*Case* 4, Gardner and Hart), or 1440° in a clockwise direction.

Considering the danger to the vascular supply, it is surprising that melæna is not a common sign. In actual fact it is very rare. It occurred in one of Gardner and Hart's cases (*Case* 20), in which severe melæna took place in a boy of 8 years. At operation there was no volvulus and bleeding was attributed to varices in the gut. Hæmatemesis was described in one of Dott's collected cases.

After unwinding the volvulus, the duodenum is examined carefully in order to ascertain the exact cause of obstruction. Ladd stresses the importance of dividing peritoneal bands which cross the duodenum from the cæcum to the posterior abdominal wall. Other authorities are equally insistent on these abnormal fixations.

That other peritoneal bands and reflections must not be neglected is clearly indicated in the case just described. Here, a second operation proved necessary on account of inadequate liberation of the first portion of the jejunum. In this case there was no abnormal fixation of the cæcum. Cathcart (1938), records a case of volvulus occurring at two days and a recurrence of symptoms at one and a half months. There was hyperfixation and constriction at the duodenojejunal flexure which required separation.

It seems possible that where a second operation is necessary, it is not due to a recurrence of the volvulus but to these accessory congenital adhesions.

The necessity for second operations opens up the question of fixation of the cæcum to prevent a recurrence of the volvulus. Most observers agree that it is of greater importance to make sure that peritoneal

bands are divided than to attempt fixation of the cæcum. In the case described in this paper, fixation of the cæcum would have been out of the question. To have forced the cæcum from the left hypochondrium, where it lay naturally, to the right iliac fossa could only have created conditions favouring intestinal obstruction. Donovan and McIntosh (1939), however, on the whole are in favour of some kind of fixation operation.

The choice of operative procedures is dependent on the actual findings. Reduction of the volvulus is sometimes all that is required. This may have to be supplemented by division of bands, fixation of the colon, or possibly some kind of short-circuit operation such as a duodenojejunostomy.

SUMMARY

The embryology of the midgut is briefly outlined. The significance of anatomical variations in relation to abnormalities in the development of this portion of the alimentary tract is briefly discussed.

A case of duodenal obstruction caused by so-called 'volvulus neonatorum' occurring in a child aged 1 year and 10 months is described in detail.

It is possible that an abnormality of rotation may pass unrecognized throughout life and death may occur from other causes. It is not known what percentage of cases pursue this course. In cases which show symptoms, the mortality-rate is comparatively high.

The high mortality is due to certain factors: First, delay in making a diagnosis. A radiograph of the abdomen is useful in demonstrating gas shadows and fluid levels. In the neonatal period, lipiodol by mouth or by means of an intranasal tube passing down the œsophagus is of great help. Barium should not be used in these cases, but can be given in older children. Barium enema gives added information.

Secondly, there is considerable risk in delaying operation after the diagnosis of duodenal obstruction has been established—there is always the danger of the occurrence of volvulus in addition to the duodenal obstruction.

Thirdly, adventitious bands and adhesions may easily be overlooked at operation and must be searched for carefully and divided after the volvulus has been dealt with.

The question of fixation of the cæcum to prevent recurrence of the volvulus is also discussed.

I wish to thank Dr. W. E. Crosbie, Superintendent of Alder Hey Children's Hospital, for permission to describe this case; Mr. W. A. Thompson for helpful suggestions and criticism; and Mr. R. B. Green and Miss Davison for the photographs and illustrations.

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PRESACRAL PERIRENAL PNEUMOGRAPHY

BY JAMES BLACKWOOD, SYDNEY

ALTHOUGH pyelography has firmly established the study of the renal pelvicaliceal systems, it seldom provides detailed information regarding the morphological outline of the kidneys themselves, or of the surrounding organs.

The possibility of demonstrating the kidneys radiologically by the perirenal insufflation of a contrast medium was first suggested when it was found that the abdominal organs were sometimes demonstrable radiologically following the induction of a pneumoperitoneum. Perirenal insufflation of oxygen as a method of rendering the kidneys and surrounding organs demonstrable radiologically, was discovered simultaneously by Carelli in Argentina, and by Rosenstein in Germany, in 1921. In this method, oxygen was introduced into the perirenal tissues via a needle inserted in the loin through the lumbocostal angle. At first this method gained considerable popularity, as it was realized that it frequently gave information unobtainable by any other method. However, it had several grave disadvantages, especially the very real danger of gas embolism. There have been several instances of serious, untoward effects from this method, including cases of fatal embolism. The danger was greatest in cases of renal neoplasm, over the surface of which there are frequently large and tortuous thin-walled vessels, and these cases are often those in which perirenal pneumography can be of most assistance. Perirenal insufflation was, therefore, usually considered to be contraindicated in such cases. Using this lumbocostal approach, perirenal insufflation was often unsatisfactory, as it was a difficult technical procedure to induce even diffusion of the gas in the correct tissue

planes. Furthermore, in cases of abnormally situated kidneys, such as in nephroptosis and renal ectopia, the standard procedure failed completely. An added disadvantage was that dual puncture was necessary if both kidneys were to be demonstrated.

Although some clinics have continued to practise this method with considerable success, its inherent dangers and disadvantages caused it to lose its initial popularity. However, fresh interest in the possibilities of perirenal pneumography was aroused by the work of Ruiz Rivas (1947, 1948) in Spain, who utilized a completely new approach. My attention was directed to this important advance by Vincent O'Connor of Chicago, to whom I owe my present interest in this field of study.

Instead of introducing oxygen directly into the perirenal tissues as in the Carelli-Rosenstein technique, air or oxygen is introduced into the loose areolar tissue in the presacral hollow immediately posterior to the rectum. A long needle, such as a lumbar puncture needle, is inserted about 1 cm. lateral to the sacro-coccygeal region in an upward, medial, and forward direction. When the end of the needle is ascertained to be in the presacral hollow, approximately 1000 c.c. of air is slowly introduced. The air is then allowed to diffuse upwards in the retroperitoneal tissues, soon surrounding both kidneys and the neighbouring structures. The diffusion of air is controlled by the positioning of the patient, following frequent fluoroscopic examinations. When the perirenal infiltration by the air is considered to be adequate, X-ray films are taken, often combined with intravenous pyelography.

Rivas claimed that this new method was safe and reliable, and that, with the even diffusion of the gas, it enabled one to obtain images, not only of the kidneys, but often also of the suprarenals, the psoas muscles, the spleen, liver, gall-bladder, and even the slips of insertion of the diaphragm.

Perirenal air insufflation by the presacral route has been performed by the present writer on fifteen occasions. The following represents the technique now adopted as a result of this experience. It is suggested that this procedure should be named presacral perirenal pneumography.

TECHNIQUE

1. Armamentarium for Perirenal Pneumography. The complete armamentarium includes a 20-c.c. luer-lok glass syringe, a 3-way stopcock with luer-lok fitting, and a 20 B.W.G. lumbar puncture needle with a 3 in. shaft and luer-lok fitting.

The use of a 20-c.c. syringe is somewhat tedious when at least 800 c.c. of air is to be injected, but it has two advantages: It is easier to manipulate than a larger type of syringe; and it also ensures that the air is injected slowly and in small amounts.

The 20 B.W.G. lumbar puncture needle has been found most satisfactory. A smaller gauge needle requires too much pressure if the air is to be injected at a reasonable rate. It is possible that with a long, thin needle, blood would not appear in the syringe when observing the very necessary precautions to obviate the danger of an intravascular injection. A larger bore needle tends to be more painful for the patient, and would theoretically be more liable to damage a blood-vessel.

The luer-lok fittings ensure stable and airtight connexions which are essential for perirenal pneumography.

2. Preparation of the Patient.—Satisfactory preparation of the patient is essential if presacral perirenal pneumography is to be undertaken. Air naturally lacks the quality of producing such intense contrast as when using a radio-opaque medium. It is especially important that intestinal gas should be reduced to a minimum.

The following routine has been found to be the most satisfactory: The evening prior to perirenal pneumography, a purgative, such as a moderate dose of cascara, is given. The sacral, perianal, and perineal areas are carefully shaved. Next morning a very light breakfast of tea and toast is given, after which the patient must fast completely. One and a half hours before the examination, a high, plain water enema is given. Half an hour prior to the examination, ½ c.c. of pitressin is injected, after which the patient is directed to indulge in moderate and normal exercise, such as casual walking. The tendency to intestinal gas appears to be increased if the patient remains in bed.

Should the patient be unduly apprehensive, it is sometimes advisable to sedate him by administering a suitable dose of morphine when the pitressin is injected.

3. Technique of Air Injection.—At first the technique of presacral air insufflation as described by Rivas was adopted. A lumbar puncture needle was inserted lateral to the sacrococcygeal joint, with the patient in the knee-elbow position. However, this technique had several disadvantages. It necessitated inserting the needle in an anterior direction in order to approach the presacral hollow, which involved a risk of perforating the rectum. The anterior direction of the needle sometimes rendered it difficult, particularly in obese patients, to be sure that its end was in the correct plane.

Another disadvantage was that it was often found to be difficult to obtain an equal distribution of the air on both sides. Sometimes the air was distributed entirely on one side, and this appeared to be especially likely when some pathological process, such as perinephritis, hindered the diffusion of air. Thus in some cases it was far easier to obtain satisfactory images of a healthy kidney than of a pathological one.

For these reasons, the technique was modified, with most encouraging results, and the following procedure is now adopted: The patient lies on his side in the lateral position with the knees and thighs fully flexed. The tip of the coccyx is palpated, and its situation noted. The area is then thoroughly cleaned with zephiran and with spirit, and draped with sterile dressing towels. The skin ½ cm. anterior to the tip of the coccyx is infiltrated with 2 c.c. of 1 per cent procaine solution using the 5-c.c. luer-lok syringe. The tissues immediately deep to the skin in this area are then infiltrated with the remaining 3 c.c. of the procaine solution. A rubber glove is put on the left hand, and the left index finger is well lubricated. This finger is gently inserted for a short distance into the rectum. The lumbar puncture needle is then introduced with the right hand ½ cm. anterior to the tip of the coccyx,

exactly in the midline (*Fig.* 157). It is gradually inserted until its tip is located in the presacral hollow by the left index finger. The needle should be introduced in a superior and slightly posterior direction. Using this technique, it is virtually impossible to perforate the rectum, especially if the position of the needle is located throughout the procedure by the left index finger.

When it has been established that the needle has been correctly placed, the 3-way tap and the 20-c.c. syringe are connected to it. Ensuring that the tap is adjusted so that the needle is connected direct to the syringe, the plunger of the latter is then firmly withdrawn. Naturally, if the slightest trace of blood is withdrawn into the syringe, the position of the needle is adjusted and the manœuvre repeated. In actual fact, in a personal series of 15 such injections, this has never been necessary. Four hundred c.c. of air is then slowly injected, care being taken that only very moderate pressure is used. The plunger is withdrawn each time to ensure that the injection will not be intravascular before the next 20 c.c. of air is injected. This rather tedious precaution obviates any slight risk of serious air embolism.

After this amount of air has been introduced, the patient remains in the same lateral position for a period of 3 minutes to enable the air to diffuse up the retroperitoneal tissues of the contralateral side to that on which the patient is lying. The patient is then instructed to turn carefully on to his other side, the needle remaining in position. Another 400 c.c. of air is injected, observing the same precautions as previously. The needle is withdrawn, and the patient remains lying on his side for a further period of 3 minutes. The injection puncture site is sealed by an application of a small quantity of collodion.

4. Radiological Technique.—Before the air injection is undertaken, a plain X-ray film of the abdomen is taken to ascertain whether the preparation of the patient has been adequate.

After the air injection has been completed, the patient is instructed to walk around for 20 minutes. Twenty-five minutes after the completion of the air injection, another anteroposterior film of the abdomen is taken. It is usually found that by this time the perirenal air insufflation is complete. Twenty c.c. of uriodone is then injected intravenously. Fifteen minutes later anteroposterior, right oblique, and left oblique films are taken. Originally, right and left lateral films were also taken, but it was found that they were seldom of much value owing to the marked superimposition of structures, and they have now been abandoned.

If it is known that there is a delayed excretion of dye in any particular patient, then the time interval following the dye injection is naturally increased, so that the films are taken at the optimum moment.

Using this standardized technique, it has been found unnecessary to observe the diffusion of air by fluoroscopy prior to radiographic examination.

Complications.—In a series of 15 presacral air injections there has not been a single instance of serious untoward effect. Two cases developed surgical emphysema of the scrotum which soon subsided without sequelæ. Another case (*Case* 6), complained of shoulder-tip pains associated with a sense of constriction in the epigastrium. This was almost certainly due to a collection of subdiaphragmatic air causing referred pains. This disappeared after 24 hours. There have been no local complications, such as sepsis or hæmatoma formation, related to the injection site. Usually the puncture hole is invisible soon after the completion of the injection, and no patient has complained of local pain or tenderness.

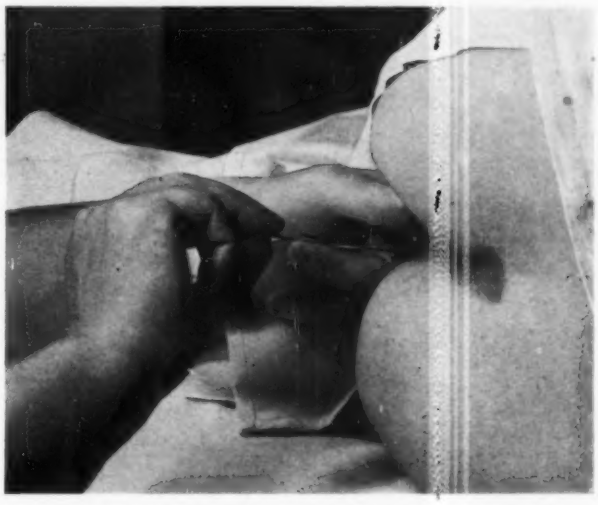

FIG. 157.—Photograph illustrating the correct method of inserting the lumbar puncture needle prior to air injection. The skin overlying the coccyx has been shaded with a skin pencil. The needle is being inserted in a superior and slightly posterior direction ½ cm. anterior to the tip of the coccyx exactly in the midline. The needle is held in the right hand whilst the left index finger has been inserted for a short distance into the rectum. The patient is lying in the lateral position with the thighs and knees flexed.

It is quite usual for the patients to feel air ascending in the retroperitoneal tissues, and this sensation is sometimes associated with a feeling of distension and discomfort which rapidly subsides after a few minutes.

CASE REPORTS

Case 1.—A male, aged 30, was admitted to hospital in June, 1950, with a history of left renal colic, frequency, and hæmaturia. Clinical examination revealed a hazy urine and tenderness in both renal angles, especially the left. Pyelography revealed an almost functionless left kidney with gross hydronephrosis and two secondary renal calculi, and a large hyperplastic, but otherwise normal right kidney.

Although routine procedures had supplied all essential clinical information, perirenal pneumography was undertaken in this case as a means of assessing the potentialities of the method. The films which were obtained were so encouraging that it was felt that the claims of Rivas and his colleagues were fully justified. The large, slightly lobulated right kidney, and the grossly hydronephrotic left kidney, were well shown. In addition, both suprarenal glands were demonstrated with great clarity, as was also the spleen (*Fig.* 158).

Case 2.—A male, aged 31, was admitted to hospital in August, 1950, complaining of pain in the left loin for 24 hours, accompanied by frank hæmaturia. He gave the family history that his father had died of polycystic kidneys, and that he had a brother and sister who were both alive and well. Clinical examination revealed gross

hæmaturia, and there was definite tenderness over the left kidney and in the left renal angle. The left kidney was just palpable, but the right kidney could not be felt. Pyelography showed abnormal pelvicaliceal systems,

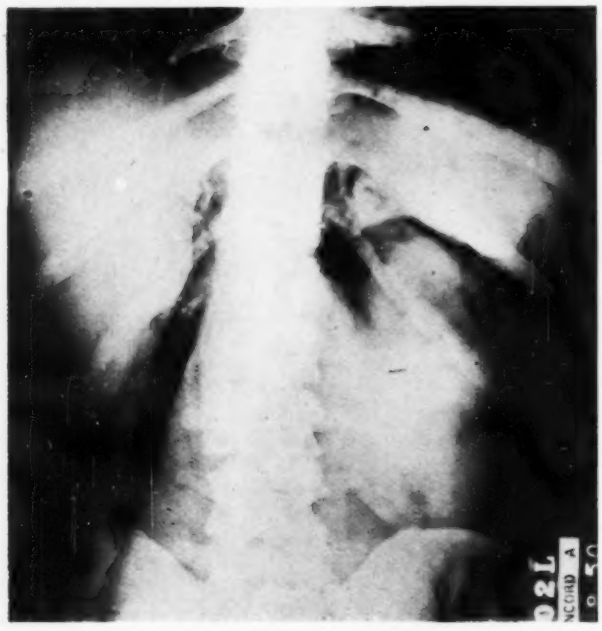

FIG. 153.—*Case* 1. Perirenal pneumogram demonstrating a hyperplastic right kidney, a hydronephrotic left kidney with two calculi, and both suprarenal glands. The spleen is also seen.

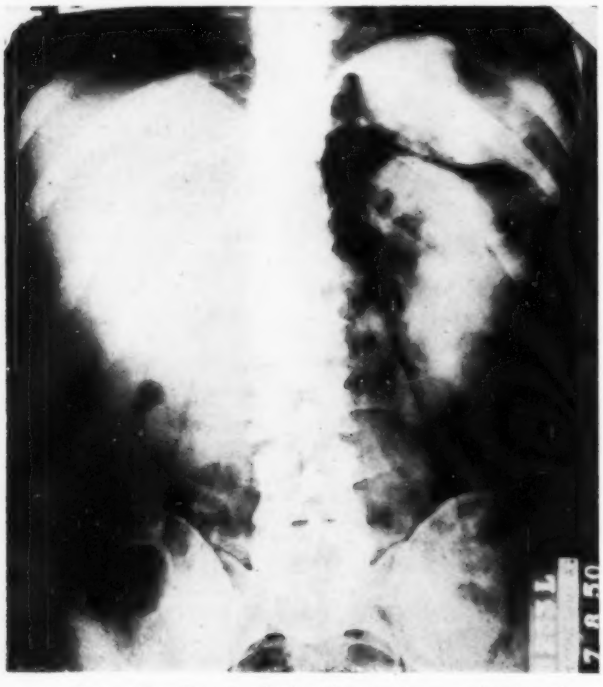

FIG. 160.—*Case* 2. Perirenal pneumogram showing the enlarged, misshapen, and irregular outline of the left kidney.

especially on the left (*Fig.* 159). There was marked distortion with compression and elongation of the major calices, associated with broad, rounded filling defects in the pelvis. There was also a small left renal calculus.

A provisional diagnosis of polycystic kidneys was made. Perirenal pneumography confirmed this diagnosis. A

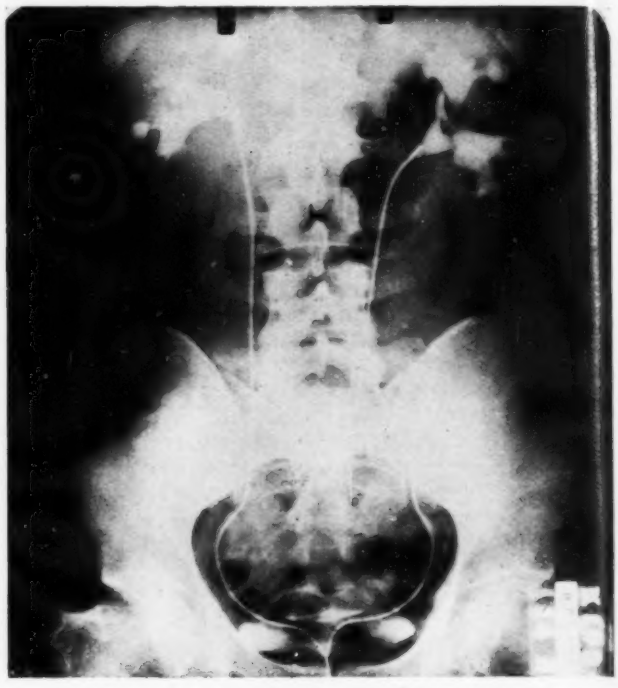

FIG. 159.—*Case* 2. Bilateral retrograde pyelograms showing abnormal pelvicaliceal system especially on the left. These changes are suggestive of polycystic kidneys.

FIG. 161.—*Case* 2. Perirenal pneumogram. Oblique lateral film confirming the diagnosis of polycystic disease of the left kidney.

grossly enlarged and misshapen left kidney was revealed, and its polycystic outline was clearly seen (*Figs.* 160, 161).

In this case the original technique of Rivas was used, and it will be seen that the air has gone entirely to the

left and that the right kidney has not been demonstrated. This difficulty to control the bilateral distribution of the air, which was encountered in several subsequent cases, provided the main justification for the modified technique which is now adopted.

Case 3.—A male, aged 51, was admitted to hospital in September, 1950, for treatment of chronic bronchitis. Whilst in hospital he developed severe hæmaturia, and he had also noticed that he had lost about 2 st. in weight during the previous six weeks. Clinical examination revealed gross hæmaturia, a moderately large prostate, and evidence of recent loss of weight. Intravenous pyelography revealed a normal right kidney, but there was no excretion of dye from the left kidney. Retrograde pyelography revealed a grossly distorted pelvicaliceal system on the left (*Fig.* 162). The right kidney appeared to be normal.

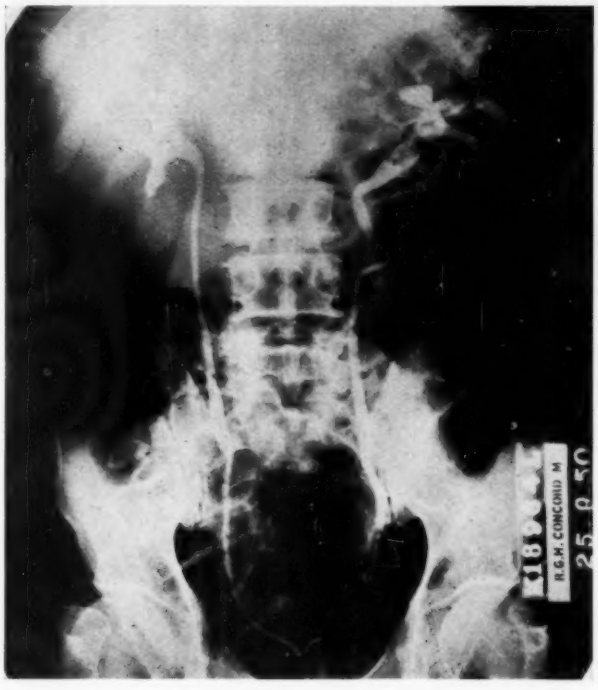

FIG. 162.—*Case* 3. Bilateral retrograde pyelograms showing the grossly distorted pelvicaliceal system of the left kidney.

Perirenal pneumography demonstrated a normal right kidney with considerable detail. The renal pedicle, the pelvis, and upper ureter, and the suprarenal gland were visualized. The left renal outline was replaced by a rounded mass, the lower pole of which was well seen (*Fig.* 163) and a diagnosis of left renal neoplasm was made. Left nephrectomy was performed by Dr. A. L. Carrodus, at which it was found that the left kidney had been almost completely replaced by the neoplasm whose rounded lower pole had been accurately depicted on the pneumogram (*Fig.* 164). Histological examination showed this to be a renal carcinoma.

Perirenal insufflation was followed by marked surgical emphysema of the left side of the scrotum which completely subsided over a period of twelve days.

Case 4.—A male, aged 53, was admitted to hospital in October, 1950, complaining of loss of weight and lassitude, associated with pains in the left hypochondrium and in both loins during the previous five months. Clinical examination revealed an obviously ill, cachectic patient. There was a large, tender, hard, superficial, bimanually palpable mass in the left hypochondrium which was emerging from under the costal margin. Intravenous pyelography revealed a hyperplastic right kidney but there was no excretion of dye from the left kidney. Retrograde pyelography showed gross distortion, compression, and lateral displacement of the left renal pelvis (*Fig.* 165). Perirenal pneumography demonstrated a hyperplastic, but otherwise normal right kidney (*Fig.* 166). The left kidney had been replaced by a very large mass whose lower pole was clearly visible (*Fig.* 167). A diagnosis of a very large left renal neoplasm was made.

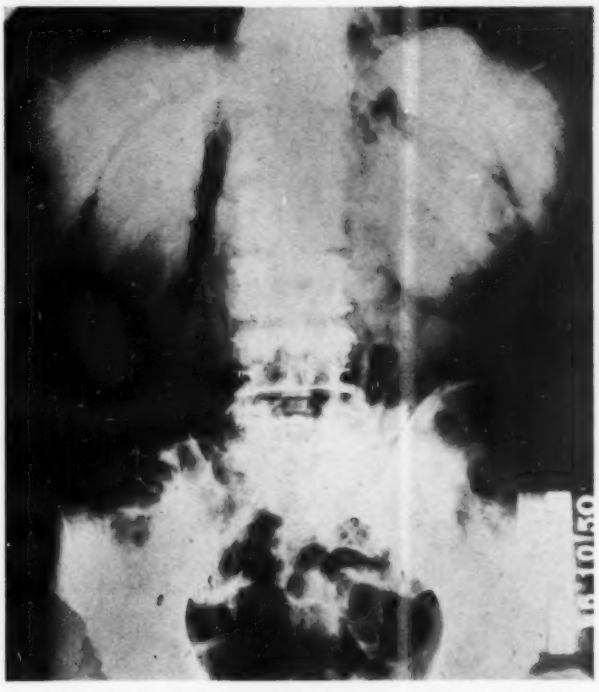

FIG. 163.—*Case* 3. Perirenal pneumogram. The right kidney is normal and has been well demonstrated. The right renal pedicle and the right upper ureter can be seen. The left kidney has been replaced by a soft tissue mass, the lower pole of which has been outlined. A diagnosis of renal neoplasm was made on these findings.

FIG. 164.—*Case* 3. Posterior view of left nephrectomy specimen. The kidney has been almost completely replaced by a renal carcinoma.

Left nephrectomy was therefore performed. A large renal mass weighing 1½ lb. was removed. The renal

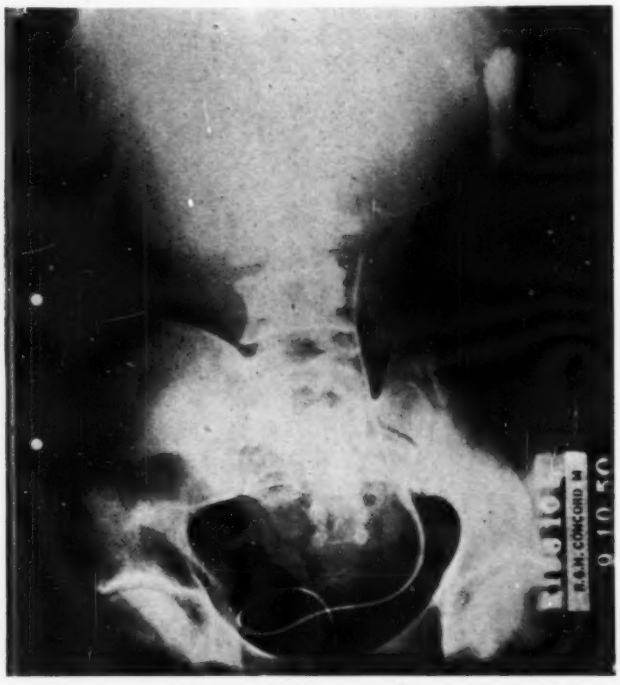

FIG. 165.—*Case* 4. Left retrograde pyelogram. There is gross distortion, compression, and lateral displacement of the left renal pelvis.

pelvis was found to be occupied completely by a large tumour which had invaded the renal cortex, and had

FIG. 166.—*Case* 4. Perirenal pneumogram. Oblique lateral film showing the slightly enlarged but otherwise normal right kidney.

extended into the perirenal tissues. Microscopical examination showed that the tumour was an undifferentiated carcinoma which had almost certainly arisen from the renal pelvis.

Case 5.—A male, aged 22, was admitted to a local hospital in September, 1950, as an acute abdominal emergency. He was suffering from severe pain in the right hypochondrium associated with vomiting. This acute attack had been preceded by occasional nagging pains in the right hypochondrium for six months. Laparotomy was performed with the provisional diagnosis of a mucocele of the gall-bladder. However, the operative findings did not confirm this diagnosis. A large cystic mass was found filling the retroperitoneal area on the right side. The exact nature of this cyst could not be

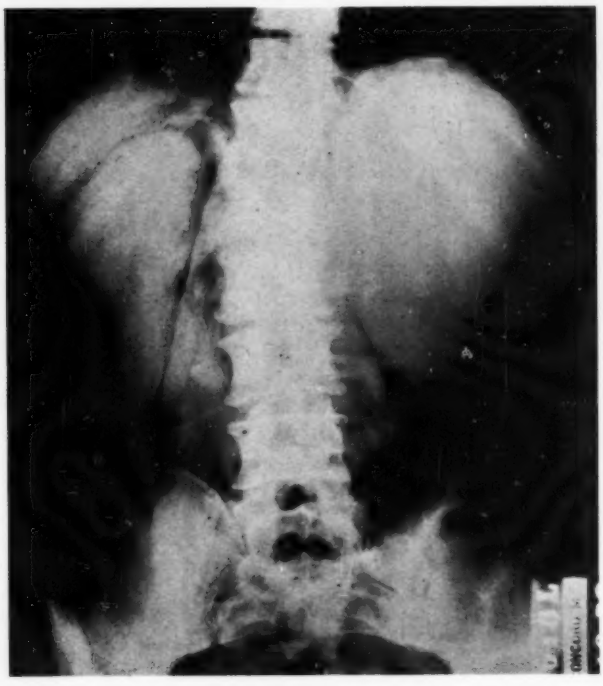

FIG. 167.—*Case* 4. Perirenal pneumogram. The right kidney is slightly enlarged but otherwise normal. The left has been replaced by a large soft tissue mass, whose lower pole can be seen. A diagnosis of a large renal neoplasm was made.

determined, so it was marsupialized to the anterior abdominal wall. A biopsy of the cyst wall showed that it consisted of dense connective tissue containing fasciculi of smooth muscle, and that it was lined by a transitional epithelium.

Immediately following this operation, large quantities of clear amber-coloured fluid were excreted via the abdominal drainage-tube. This fluid contained sodium chloride and urea, but had a negligible amylase content. This fluid amounted to over 70 oz. a day, and the patient soon developed marked polydipsia. He was transferred to Sydney, where he finally came under the care of Dr. A. L. Carrodus. Intravenous pyelography revealed a normal left kidney, but there was a well-marked right hydronephrosis. This was confirmed by retrograde pyelography. The indigo-carmine test was performed, and no dye issued from either the right ureteric orifice or from the abdominal drainage-tube. The right kidney urine contained pus cells and grew *Pseudomonas pyocyanea* on culture.

Perirenal pneumography was undertaken, combined with the simultaneous injection of 25 c.c. of uriodone into the drainage-tube, which had been firmly clamped. This procedure revealed a normal left kidney and suprarenal, together with a gross hydronephrosis on the right side (*Fig.* 168). The uriodone had filled the dilated pelvicaliceal

system and the perirenal pneumogram revealed the hydronephrotic renal outline. This confirmed that the retroperitoneal cystic mass which had been marsupialized was actually a large hydronephrotic sac.

Case 6.—A female, aged 47, was ad[illegible]ted to hospital in October, 1950. She had noticed a[illegible]wly enlarging mass below the left costal margin for [illegible] months. She complained of no other symptoms exce[illegible]r some recent

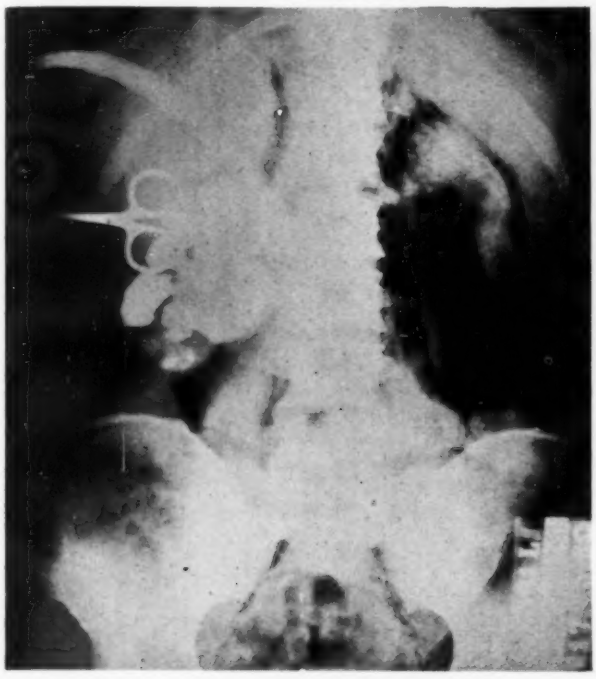

FIG. 168.—*Case* 5. Perirenal pneumogram. The left kidney and left suprarenal appear normal. The right kidney is represented by a hydronephrotic sac, which has been filled with uriodone by injection through the abdominal drainage tube. The much thinned cortical tissue of the lower pole is well seen.

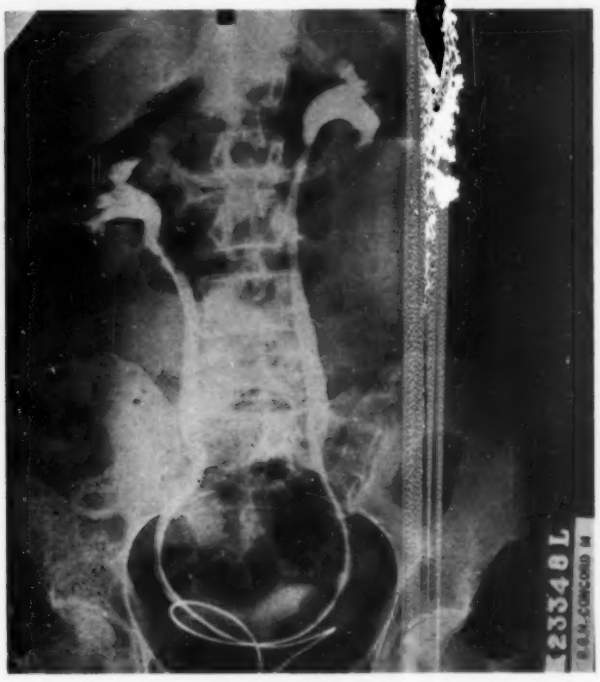

FIG. 169.—*Case* 6. Bilateral retrograde pyelograms. Both pelvicaliceal systems appear normal except that there is some medial displacement of the upper third of the left ureter and the left renal pelvis appears slightly elevated. There is also a soft tissue mass which appears to be related to the lower pole of the left kidney.

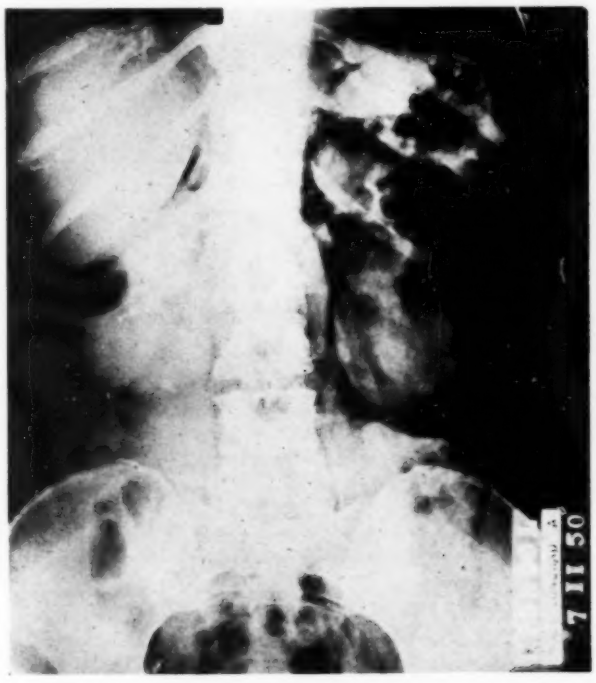

FIG. 170.—*Case* 6. Perirenal pneumogram. There is a large mass attached to the lower pole of the left kidney, although there is no distortion of the pelvicaliceal outline. The left suprarenal gland can also be seen and appears normal.

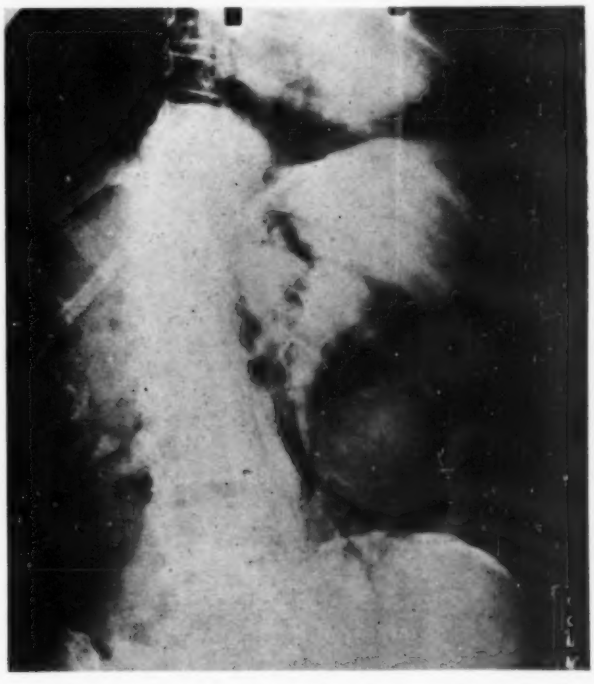

FIG. 171.—*Case* 6. Perirenal pneumogram. Oblique lateral film showing a large mass attached to the lower pole of the left kidney which has destroyed the adjacent renal tissue. A diagnosis of a left renal cyst or neoplasm was made on these findings.

loss of weight. There had been no urinary symptoms whatever. Clinical examination revealed a mobile kidney. Retrograde pyelography confirmed these findings, but provided no additional information (*Fig.* 169).

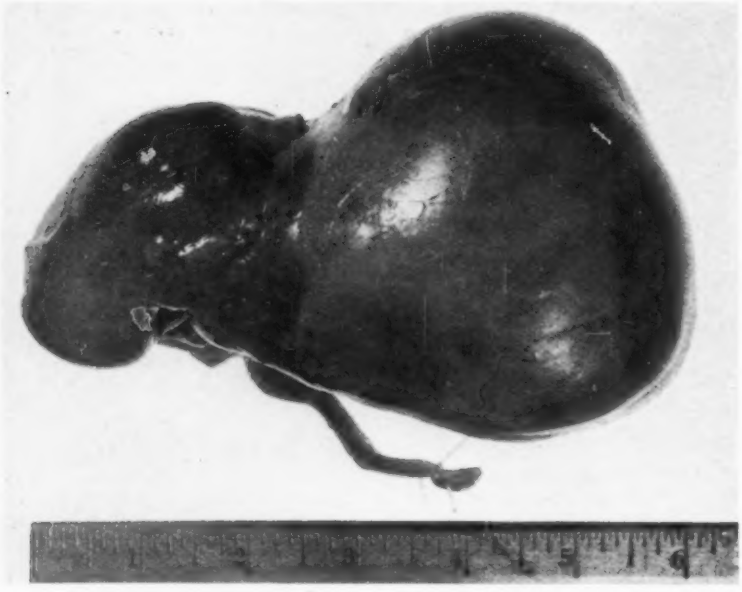

FIG. 172.—*Case* 6. Left nephrectomy specimen. There is a large, smooth mass attached to the lower pole of the kidney, the anatomy of which had been demonstrated by perirenal pneumography. Histological examination proved this mass to be a renal carcinoma.

Perirenal pneumography proved to be invaluable in this case. It revealed that here was a large, rounded mass arising from the lower pole of the left kidney which had destroyed the adjacent renal tissue (*Figs.* 170, 171).

Left nephrectomy was performed by Dr. A. L. Carrodus, at which this mass was found to be a renal carcinoma (*Fig.* 172). This was confirmed histologically. Perirenal pneumography in this case provided information which was unobtainable by pyelography, and which was essential if the tentative diagnosis of a renal mass was to be confirmed.

Case 7.—A male, aged 47, had been diagnosed eight years previously as suffering from hypertension. He was complaining of increasing lassitude and dyspnœa of effort. His B.P. was 220/110 mm. and his urine contained albumin. Abdominal examination revealed a nodular mass in each loin which was bimanually palpable, and a tentative diagnosis of polycystic kidneys was made, and this was confirmed by intravenous pyelography.

Perirenal pneumography demonstrated huge bilateral polycystic kidneys (*Figs.* 173, 174).

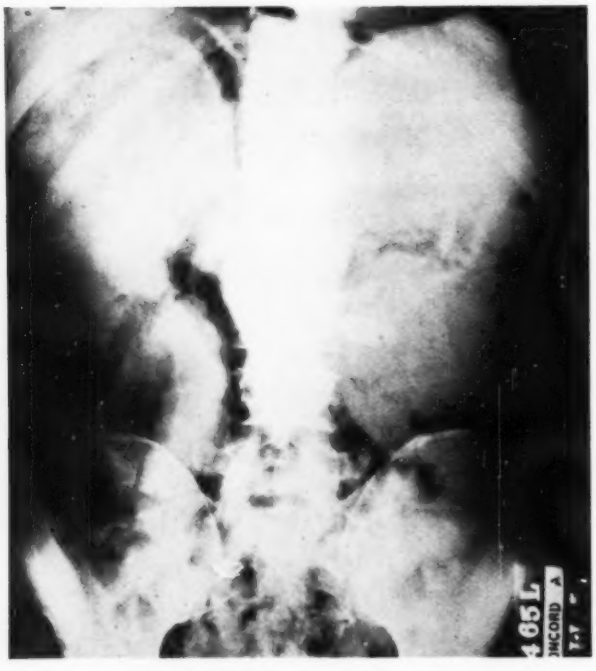

FIG. 173.—*Case* 7. Perirenal pneumogram showing huge polycystic kidneys.

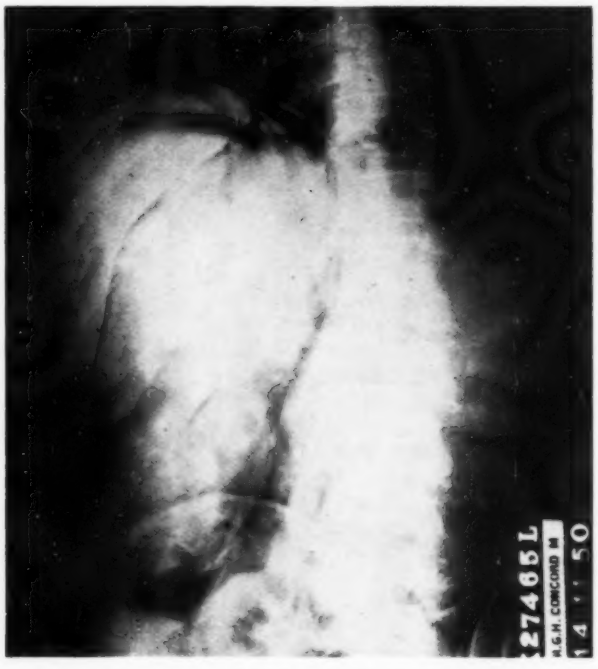

FIG. 174.—*Case* 7. Perirenal pneumogram. Oblique lateral film showing the much enlarged, polycystic left kidney.

discrete mass in the left hypochondrium which was bimanually palpable from the loin. There were no other physical signs, and examination of the urine revealed no abnormality. Intravenous pyelography showed no abnormality of the renal caliceal outlines, but there was some medial displacement of the upper third of the left ureter, and there was a soft tissue mass which appeared to be related to the lower pole of the left

DISCUSSION

Presacral perirenal pneumography has proved itself to be the most successful clinical method of demonstrating the anatomy of the kidneys and surrounding organs. It frequently provides information which is unobtainable by any other method. In many lesions of the kidneys, pyelography may

appear normal or equivocable. Formerly in these cases one either had to accept tentative diagnoses based on clinical, pathological, and pyelographic findings, or one resorted to operative exploration. Now with the aid of perirenal pneumography, one is nearly always able to form a confident diagnosis. This is especially true, of course, where there is a marked distortion of the renal outline.

Perirenal pneumography should prove invaluable in the diagnosis of suprarenal tumours. It should prove most informative in cases of small tumours, such as pheochromocytomata, which cannot be demonstrated by any other method.

The usefulness of perirenal pneumography as a diagnostic measure has been known for nearly thirty years, but hitherto there has been no reliable and safe method. The danger of air embolism and the frequency with which the procedure failed, resulted in it being abandoned by most workers. Perirenal insufflation by the presacral route appears to be both safe and reliable. If routine precautions are observed, it would seem that the danger of air embolism is remote, especially as the most highly vascular area traversed is the skin. Using the present standardized technique, it is seldom that one fails to demonstrate the kidneys and suprarenals satisfactorily. An added advantage is that the single injection demonstrates both kidneys simultaneously, thus eliminating the need for dual puncture.

Radiological follow-up has shown that the injected air is usually absorbed after ten days, when the procedure can be repeated if necessary. Air is a satisfactory contrast medium and there would appear to be little advantage in using oxygen instead. The use of a manometer also seems unnecessary, as one naturally ensures that the air is injected slowly and at a low pressure.

Air readily tracks up the retroperitoneal tissues from the presacral area and the perirenal emphysema is often complete within a few minutes. When the air reaches the lower part of the kidney, it usually diffuses in an even manner over its anterior and posterior surfaces. There does not appear to be any anatomical barrier to this even perirenal diffusion. The upward filling of the perirenal space commencing from the lower pole of the kidney is often demonstrated very clearly on the lateral oblique films. (*Figs.* 161, 166.)

It is difficult to reconcile these findings with the recent work of Mitchell (1950), who has investigated the anatomy of the perirenal fascia and perirenal space. His experimental method was to observe the fate of a radio-opaque solution when injected into the perirenal space via the lumbar route. His findings were based on subsequent radiological and anatomical studies. He concluded that the anterior and posterior layers of the perirenal fascia are fused all around each kidney. In other words, he claims that the perirenal space is a closed one. These findings are at variance with the views of the earlier workers such as Zuckerkandl (1883), and Sappey (1889). They affirmed that the two layers of the perirenal fascia were fused superiorly and laterally, but were not fused medially or inferiorly. They visualized the perirenal fascia and space to be open inferiorly like an inverted sac.

Perirenal pneumography tends to show that the truth may be halfway between these two divergent views. It would seem that the perirenal space is certainly closed laterally and superiorly, and also medially. In most cases in which the air has been injected on one side only, there has been no tendency for it to cross the midline once it has ascended to the renal areas. Also, in only one instance has air tracked superiorly to reach the thorax, and then in only a small amount. The clarity with which the suprarenal glands are usually demonstrated confirms the belief that the perirenal fascia invests both the kidney and the suprarenal. However, the ease with which injected air will fill the perirenal space from below would support the view that the space is in effect an open one inferiorly. There is no doubt that further study of the anatomy of the perirenal tissues is indicated.

It is my belief that presacral perirenal pneumography will soon become an accepted and much practised method of investigation. It is safe, reliable and informative. The advent of pyelography rendered surgical exploration of the loin an unusual diagnostic procedure. Presacral perirenal pneumography should almost eliminate the necessity for such operative methods in the diagnosis of renal and suprarenal pathology.

SUMMARY

1. The radiological demonstration of the kidneys and surrounding organs by the perirenal insufflation of gas is discussed.

2. A new method of perirenal air insufflation is described.

3. It is suggested that the name of presacral perirenal pneumography be given to this procedure.

4. Illustrative cases demonstrating the value of this new technique are described.

5. It is claimed that this method is safe and reliable and a definite advance as compared with previous techniques.

6. The information gained by presacral perirenal pneumography regarding the anatomy of the perirenal space is briefly contrasted with the findings of recent workers.

It is a great pleasure to acknowledge my indebtedness to the many people who have assisted me in the preparation of this paper. I am especially grateful to Dr. A. L. Carrodus for his assistance and encouragement; to Dr. J. Colclough and his radiological team for their invaluable co-operation; to Miss Dorothy Lee for the excellent photographic reproductions; and above all to Dr. Vincent O'Connor, of Chicago, who first aroused my active interest in perirenal pneumography.

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MEDIASTINAL GOITRE

By EDRIC WILSON, Plymouth

Enlargement of the thyroid gland so that a portion of it lies below the plane of the superior thoracic inlet is relatively common in areas where goitre is endemic; in my own series of over 800 thyroidectomies, drawn from Devon and Cornwall, the incidence is approximately 12 per cent.

This type of enlargement is usually known as substernal or intrathoracic, but Sweet (1949) differentiates between the substernal type, in which there is a large component in the neck, and the intrathoracic, where the largest diameter lies below the thoracic inlet. Wakeley and Mulvaney (1940) discuss the variations of the frequency of this condition which has arisen from the different methods of classification. They regard the classification into partial and total intrathoracic as too indefinite and confine their described cases solely to swellings mainly or wholly within the thorax. Rives (1947) prefers to classify them as substernal, lying in the anterior compartment, in front of the trachea; and the "mediastinal aberrant", which does not lie in the classical location.

Mediastinal Aberrant Goitre.—It is difficult to understand why the term 'aberrant' has been chosen. An aberrant thyroid may be defined as an enlargement which has wandered away from the cervical thyroid or that it has no connexion with the gland or its blood-supply. This term could be used to include such varieties as thyroid tissue lying on the diaphragm and not connected with the thyroid. Wakeley and Mulvaney (1940) say that the possibility of such aberrant growth is doubtful but feasible, as in the early stages of development the thyroideal bud lies in front of the second aortic arch, coming later into contact with the aortic stem in the loose subpharyngeal tissues below the arches. The possibility of such aberrant or ectopic growth is a real one but, so far, search of the literature has not revealed a case which could be explained only on these facts. Keynes (1950), in following Rives's classification, describes a case in which there was no connexion with the thyroid gland but a very large vascular pedicle turned down from the right thyroid axis, and two venous channels appeared to pierce the pericardium and empty direct into the auricle. This abnormal venous return might be explained by some failure of the normal arrangement whereby the inferior thyroid veins empty into the innominate vein.

It would seem that this classification has little practical value, and an analysis of the cases quoted by Rives shows the diversity of the pattern.

The following sixteen are all the cases that I could find in the literature classified as 'mediastinal aberrant'. Histological details are lacking in some of these cases, but the tumour has been assumed to be thyroid tissue.

Rives describes three personal cases and quotes cases cited by Mora, Berard, and Berard (1939), Von Haberer (1938), Roholm (1938), Urban (1939) two cases, Henschen (1940), Hicken (1936), Hollenberg (1946), Eggers (1941), De Courcy and Price (1944), Mason (1942), Cabot (1936), and Soley and Rinehart (1939).

After reviewing these cases it appears that they are a mixed group, many of which are incompletely reported, and I do not feel that there is any advantage in the adoption of the term 'mediastinal aberrant'.

Suggested Classification. — It would seem simpler and more helpful in the operative treatment to classify these enlargements on anatomical grounds as anterior and posterior mediastinal. The anterior mediastinal goitre comprises the larger number. It lies in front of the trachea, great vessels, and recurrent nerves, and is usually connected to the thyroid proper by a wide pedicle. The posterior mediastinal goitre lies posterior to any of the following structures: the recurrent nerve, inferior thyroid artery, the carotid sheath, innominate and subclavian arteries, innominate veins, and on the right side the superior vena cava. It lies against the vertebral column and tends to press the œsophagus laterally, and the trachea is often displaced forwards. The pedicle attaching it to the gland is frequently small and may consist of vessels within a fine sheath of areolar tissue.

As it seems likely that more cases of the posterior type will be discovered, the importance of an anatomical classification is apparent. Where it is known that the tumour is in the posterior position the operator is aware of the greater danger to the large veins and recurrent nerves, and preparation for a more extensive exposure can be made. The anterior mediastinal goitre needs little discussion. Removal through a cervical incision is usually easy unless the goitre is very large, when it may be necessary either to split the sternum or break into the centre of the mass and evacuate the contents to reduce the size. This latter step should be avoided as the possibility of carcinoma is always present; for example, in one of my cases the intrathoracic portion, although showing areas of calcification, on microscopical examination proved to be malignant. In this connexion Keynes, in quoting the case of posterior mediastinal goitre described by Mora, Isaacs, Spencer, and Ediden (1944), states that the post-operative X-ray film suggested that a part of the tumour was still in the thorax, and from examination of the illustration this certainly seems to be the case. In the description of the operation the mass had been broken into in the course of removal through a cervical incision and may have been incompletely removed.

Goitres in the Posterior Mediastinum.—The posterior mediastinal goitre is uncommon; 12 cases have been described which can be placed in this group, and two new cases are recorded.

Since this paper was written J. S. Tomkinson (1951) has described 3 cases and mentions that Linnell and Piercy (1949) have encountered several.

Of the cases quoted by Rives, mentioned above, those of Von Haberer, Roholm, and Mason can be classified as posterior mediastinal, as can also one of Rives's cases.

The cases described by the Berards, Urban, and Reufer might possibly be included, and are accepted by Mora, but for lack of accurate description they are omitted here.

Sweet (1949) describes 6 cases, but the first two do not appear to have descended into the thorax.

Keynes describes 2 cases. In the first there was a pear-shaped shadow in the upper part of the right

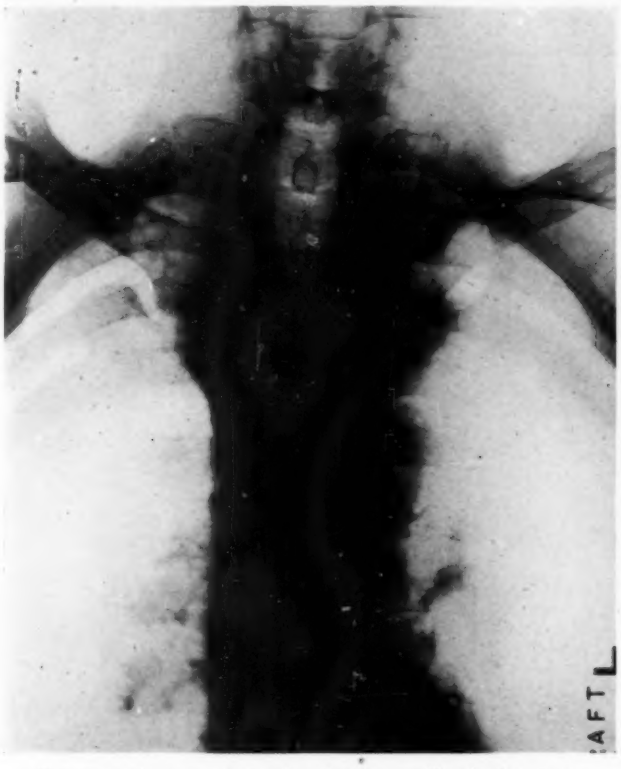


FIG. 175.—Anteroposterior view showing swelling in the upper mediastinum.

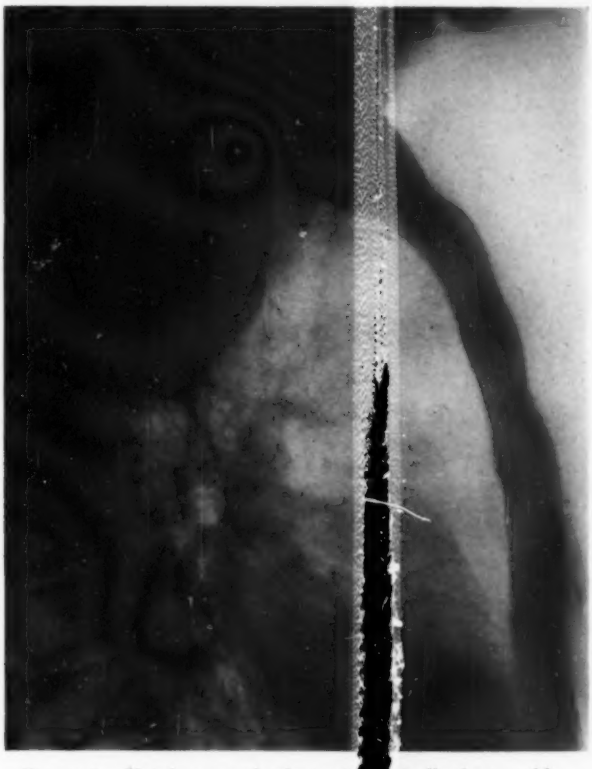


FIG. 176.—Showing mass in the poste[illegible] mediastinum, with trachea anterior to [illegible]

posterior mediastinum behind the trachea, which was displaced with the œsophagus and aortic arch to the left. In the second case the lateral view showed extreme displacement of the trachea which was pushed far forward by the tumour. A thyroid enlargement was found on the left side of the neck. The tumour was behind the left innominate vein, and weighed 290 g.

D. B. Wightman and H. M. Windsor (1951) describe a case producing mainly pressure symptoms. A large adenoma weighing 254 g. was found to the left and posterior to the trachea.

CASE REPORTS

The details of my own cases are as follows :—

A. L., male, aged 61, attended hospital on Oct. 9, 1950, complaining of fits of depression and feeling " all in ". For six months he had had intermittent pain in the left chest, with no relation to exertion. He had lost weight and was 7 st. 3 lb. X-ray examination showed " a large, oval, sharply defined shadow in the upper posterior mediastinum pushing the trachea forward and to the right. It does not suggest malignancy " (*Figs.* 175, 176). Wassermann negative. Pulse 100 per minute. B.P. 180/110. He was admitted for further investigation on Oct. 31. A barium swallow (*Figs.* 177, 178) " confirms a large posterior mediastinal tumour displacing the œsophagus forwards and to the right. It appears to extend from the second to the seventh dorsal vertebræ. No bony changes are seen in the upper dorsal and cervical vertebræ ". On further examination a swelling in the base of the right side of the neck was found and considered to be a nodular thyroid. With Keynes' description in mind it was decided that this was a posterior mediastinal goitre. It appeared that some of his symptoms were those of thyrotoxicosis, so he was given I[illegible]l's Iodine, 5 min., t.i.d., for six days.

AT OPERATION, Dec. 1.—Intrat[illegible]neal nitrous oxide and oxygen. Collar incision. An ex[illegible]sion of the thyroid gland below the left sternoclavicu[illegible] joint was found. The superior thyroid arteries on [illegible]n sides were tied; the inferior thyroid arteries were t[illegible] under the carotid sheath, and the right lobe dislo[illegible]ed and subtotally removed. The anterior surface of t[illegible] mass was explored with the finger, but it was not possib[illegible] to reach the lower pole completely. Since a large ve[illegible] might have been stretched over it, the use of any forc[illegible] seemed dangerous. The sterum was split vertically and [illegible]ral incisions were made to the second space, and a go[illegible] exposure was obtained. It was then found that alth[illegible]gh the mediastinal mass was in contact with the lower p[illegible]e of the left lobe of the gland, there was a groove dividi[illegible] the two structures. The left innominate vein was fou[illegible] stretched over the anterior surface of the lower part of t[illegible]e mass, which itself was adherent to the left side of the trachea. Both here and posteriorly it was more difficul[illegible] to find the easy plane of cleavage usually found in removi[illegible] an anterior mediastinal goitre. The recurrent laryn[illegible]al nerves on both sides were identified and preserv[illegible]. The left carotid artery was seen to be displaced to the left, the trachea, and œsophagus partly to the right and anteriorly, and the vertebral bodies were seen posteriorly. A large branch of the left inferior thyroid artery entered the posterior surface of the upper pole of the tumour, which was a

flattened oval 4 ins. (10 cm.) in length (*Fig.* 179), weighing 4 oz. (111 g.). The thyroid proper (*Fig.* 180) weighed 1¼ oz. (33·5 g.). The post-operative course was uneventful.

Another case came under the care of my colleague, Mr. C. F. Mayne, and through his courtesy I am able to give the following details :—

FIG. 177.—Barium swallow showing displacement of the œsophagus forwards.

FIG. 178.—Showing displacement of the œsophagus to the right.

FIG. 179.—Posterior mediastinal goitre with a branch of the left inferior thyroid artery entering the upper pole. (× ⅔.)

FIG. 180.—The thyroid proper, showing some nodularity (× ⅔.)

PATHOLOGICAL FINDINGS.—The mediastinal mass is composed of thyroid tissue, partly active and partly resting. The thyroid gland is composed mainly of atrophic and resting tissue.

When seen as an out-patient at the end of January he stated that he felt well and was anxious to return to work soon. The pulse-rate was 80 and the weight 7 st. 3 lb.

E. R., male, aged 72, complained of a swelling in the right side of the neck, present for two months, which was painful at times. He was a healthy looking man and said he could walk for miles, and made no complaint of respiratory difficulty. On examination there was a large swelling of the right lobe of the thyroid, with distension of the external jugular veins. The lump moved

easily on swallowing and it was possible to put a finger under the outer side of the lower pole. Radiography of chest (*Figs.* 181, 182) show a " Large upper mediastinal shadow displacing the trachea to the left. This mass does not appear to be in the anterior mediastinum ".

FIG. 181.—Enlarged upper mediastinal shadow displacing trachea to the left.

FIG. 182.—Lateral view showing posterior mediastinal goitre.

Barium swallow (*Fig.* 183): " Barium passes easily down the œsophagus. The mass appears to be displacing the œsophagus to the left and only slightly forwards, and seems to be in the middle mediastinum encroaching on the posterior mediastinum. Suggests possibly glandular enlargement, but the mass moves on swallowing."

As the patient's symptoms were so slight and he appears otherwise to be in good health, it was felt that operation should not be performed, but he has been advised that if there is any increase in the size of the swelling, or difficulty in breathing, he should immediately seek advice.

Since this article was accepted for publication another case has come under my care.

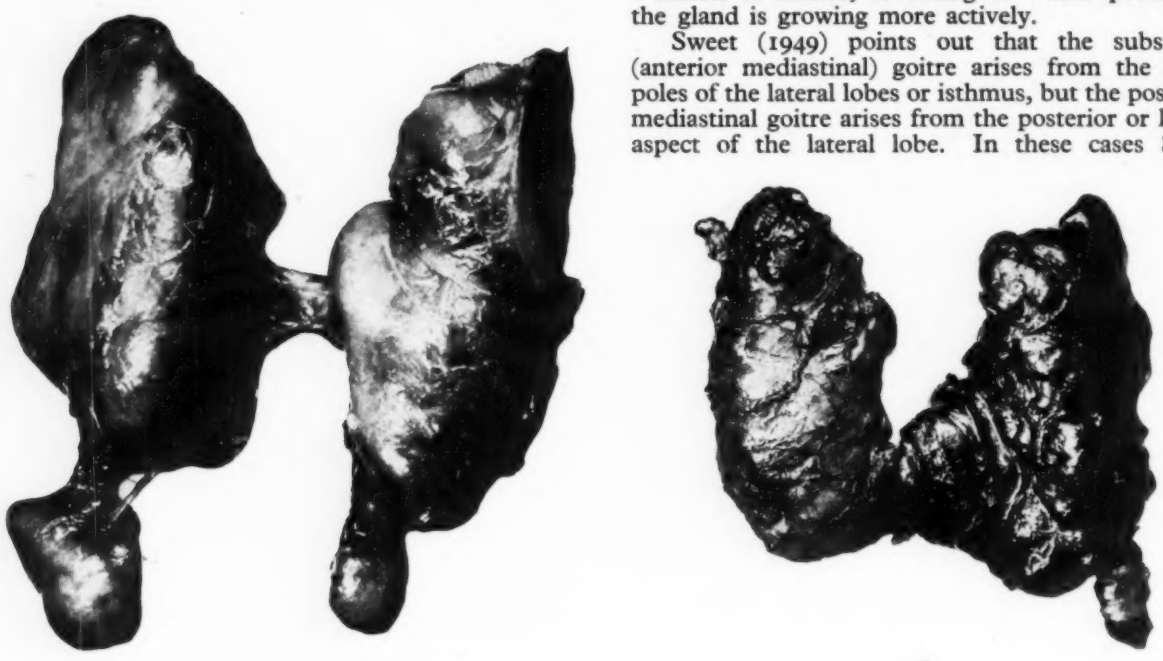
A. E., female, aged 57, had an enlarged thyroid for twenty years, and recently had become breathless on exertion, with œdema of the ankles at times. Her weight was stationary at 14 st. The skin was moist, and the pulse-rate was 88.

Examination of the thyroid showed gross enlargement of the central and both lateral lobes. The face became very congested on raising the arms above the head. Radiography showed an enlargement of the normal mediastinal shadow on the right side, which extended upwards towards the thoracic inlet. The aortic knuckle was displaced to the left. The lateral radiograph was unsatisfactory owing to the obesity of the patient.

At operation the right lobe was found to extend below the clavicle, with the right common carotid artery anterior to it. The right recurrent laryngeal nerve was identified and preserved, and a very large right inferior thyroid artery was tied beneath the carotid sheath.

FIG. 183.—Showing displacement of the œsophagus to the left.

Further dissection showed the impossibility of removing the mass without division of the sternum; this was done and the innominate artery was seen to cross the mass, indenting it deeply. The lower pole was adherent to the surrounding structures and needed careful dissection. The difficulty of this dissection contrasted strongly with the ease of enucleation of the anterior mediastinal goitre.



A B

FIG. 184.—A, Showing early stage of bilateral mediastinal goitre; B, Showing development of solitary nodule from posterolateral aspect of gland.

The mass was soft and had been broken into at one point, but was removed essentially intact. Had the mass been removed piecemeal it would have been difficult to identify the correct plane of cleavage, with a consequent risk of incomplete removal.

The left lobe of the thyroid weighed 194 g., and the right lobe 178 g. The mediastinal portion weighed 102 g., and microscopically showed mainly colloid change, with no evidence of malignancy.

THEORIES OF CAUSATION OF INTRATHORACIC GOITRE

Many explanations for the migration of thyroid tissue into the thorax have been put forward, among them breathing, swallowing, gravity, and muscular pressure, forcing the more solid structure into the pliant tissues of the thoracic inlet. There may be some cases in which an embryological explanation may be satisfactory, but the other hypotheses seem unnecessary if the manner of growth of large nodular goitres is considered. It is not unusual to find a large nodule in the superior pole behind the superior thyroid vessels, extending upwards towards the angle of the jaw; or a similar mass growing from the isthmus and, although covered by the pretracheal muscles, projecting well forwards. Similarly, a large nodule may be found to have inserted itself between the trachea and cervical œsophagus or to have enlarged laterally and displaced the carotid vessels. In a few cases of primary toxic goitre in young women I have been able to demonstrate a strap-like extension, which is often bilateral, downwards from the lower part of the lateral lobe, about ¼ in. (7 mm.) in width and terminating in a slight enlargement (*Fig.* 184). Such extensions might well develop into anterior mediastinal goitres. From this it would seem that the direction of enlargement is a matter of chance, according to which portion of the gland is growing more actively.

Sweet (1949) points out that the substernal (anterior mediastinal) goitre arises from the lower poles of the lateral lobes or isthmus, but the posterior mediastinal goitre arises from the posterior or lateral aspect of the lateral lobe. In these cases Sweet states that the posteriorly descended goitres pass downwards through a space bounded laterally by the first rib, posteriorly by the vertebral column, medially by the trachea and œsophagus, and anteriorly by the carotid sheath.

INDICATIONS FOR OPERATION

It is very probable that the condition is not so uncommon as has been supposed, and with the more frequent use of mass radiography, followed up in suspicious cases by lateral views of the chest and opaque meals to define the position of the œsophagus, many more examples will be discovered. When a mediastinal mass is discovered examination of the neck may reveal only a very small nodule in the thyroid, which indicates that the gland is abnormal and that therefore the mediastinal mass may be of thyroid origin. The indications for operation should be discussed. It may seem unnecessary to advise operation where the tumour is discovered accidentally, but it must be borne in mind that any increase in size makes operation technically more difficult. The degenerating or cystic goitre is likely to undergo sudden increases in size which may lead to dangerous pressure symptoms. Thyrotoxicosis is an indication for operation. The toxic symptoms may be overlooked because of the more obvious mechanical disturbances, and operation performed without preliminary treatment may produce dangerous reactions.

Since many of these patients are elderly it should be remembered that the presenting symptoms in them differ considerably from the classical picture in the young. The symptoms of toxicity in these cases, such as tachycardia, sweating, tremor, emotional instability, and the eye changes, are not usually so marked, and only one symptom may be present. The cervical goitre is often shrunken, and the most careful examination may reveal only the presence of what seems to be a very small nodule, and on removal the gland is found to be two or three times larger than normal.

In the author's case, except for the unstable pulse, the clinical picture could easily have been mistaken for one of the depressive states.

Wakeley and Mulvaney point out that there is little reference in the literature to the toxic manifestations produced by mediastinal goitres; in their series of 20 cases, 7 were clinically thyrotoxic.

OPERATIVE APPROACH

The value of an anatomical classification becomes apparent when the operative approach to the posterior type is considered. When it is known that the tumour is in the posterior position the operator is aware of the greater danger to the large veins and recurrent nerves. The method of exposure has varied; the thyroid surgeon prefers the cervical incision with splitting the sternum, if necessary; the thoracic surgeon often uses a purely thoracic approach. It is interesting to review the methods used in the recorded cases. In Sweet's *Case* 3 removal through a cervical incision failed, whereupon a " standard thoracotomy through the fourth space " was done, but a medial extension in the region of the trachea and œsophagus made it necessary to re-open the cervical incision and deliver the mass by that route.

Keynes, from his large experience of anterior mediastinotomy, strongly favours the cervical approach, with sternal splitting if necessary. The advantages of this are that the thyroid gland is easily and fully exposed, the inferior thyroid arteries are ligated well away from the gland, and the ' gossamer-like ' plane of cleavage around the gland is entered and can be followed around the intrathoracic enlargement. If thought advisable, the sternum is split as far as necessary, the finger re-enters the plane of cleavage, and the structures anterior to the tumour are displaced or divided as necessary. It is then possible to dislocate the tumour forwards and free it from the trachea and recurrent nerve under good vision. Splitting the sternum should not entail opening the pleural cavity, which, although not so dangerous as it used to be, may cause some complications in convalescence. The discomfort of a split sternum properly repaired is minimal, and the manœuvre is quickly performed.

SUMMARY

1. Recorded cases of so-called aberrant mediastinal and posterior mediastinal goitres have been reviewed.

2. A classification of intrathoracic goitres on anatomical lines into anterior and posterior mediastinal is put forward.

3. The evolution of intrathoracic goitres is discussed.

4. Anterior mediastinal goitres are relatively common and present few operative problems. So far as can be ascertained the posterior mediastinal goitre is uncommon; 12 cases are mentioned and 3 new cases recorded.

5. The symptomatology is discussed.

6. Operative approach through a cervical incision and anterior mediastinotomy, if necessary, seems preferable.

I wish to thank Dr. S. G. Galstaun and Dr. J. Cowen for the X-ray findings; and Dr. J. O'Brien for much assistance and for the report on the specimens; and Miss J. Butler for the photographs and prints.

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ORBITAL COMPLICATIONS OF HEAD INJURY

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WITH the increased speed of modern transportation the head-on impact has become one of the commonest results of road and air accidents. Although these impacts produce injuries of the facial skeleton, the eye, and the cranium, the scope of the present paper is limited to those cases in which the disturbance within the orbital structures may be considered a complication of the craniocerebral injury. In some cases the relevant importance of this complication is slight, as in the transient diplopia which follows many head injuries, while in others the orbital complications may be considered the major disability.

The early observation and detection of these complications is of importance when it is realized that blindness in one eye is rated by Insurance actuaries as equal in value to the loss of a limb. In spite of this fact, these complications appear to be often overlooked. Usually the omission is due to the difficulty of detection of a visual defect in an unconscious or unco-operative patient, or to the ecchymosis and swelling of the eyelids which may mask gross structural derangement of the orbit. Nevertheless, it must still be admitted that in many cases there has been a failure to recognize signs indicating a lesion in the neighbourhood.

Orbital complications are made manifest by a triad of signs and symptoms which is not difficult to detect in a reasonably co-operative patient. The primary need is for the observation of these signs; diagnosis and treatment depend upon the interpretation of these signs. For this reason, it is proposed to discuss the orbital complications of head injury in relation to the symptoms and signs which are produced and to attempt to correlate the clinical findings with the known facts regarding the exact causation of these disturbances. Ocular signs of increased intracranial pressure, such as papillœdema and pupillary dilatation, will not be considered within the scope of this paper.

In this series of 58 cases, occurring in approximately 500 personally observed cases of head injury, some disturbance of visual function proved a major complication. The clinical features of these complications fall into three groups: deterioration or loss of vision in one or both eyes, double vision, and displacement of the eye. The components of this triad were found in the following proportion :—

Deterioration of vision	33 cases (57 per cent)
Double vision	19 cases (33 per cent)
Displacement of the globe	19 cases (33 per cent)

DETERIORATION OF VISION

A wide variety of visual defects fall within this group, defects which vary in degree from blurring of vision sufficient to preclude reading large print in a newspaper to complete loss of vision, or even loss of the eye itself. In this series, which is composed primarily of craniocerebral injuries, lesions such as commotio retinæ, rupture of the choroid, or traumatic chorioretinitis, were not observed. The lesions which were identified as causing the deterioration of vision fall into three separate groups: (1) neuromuscular paralysis with loss of accommodation; (2) intra-ocular hæmorrhage; and (3) injuries to the optic nerve or chiasm or to the blood-supply of these structures.

Blurred Vision.—This was the main complaint in 10 patients. The obvious defect in these cases was a paralysis of accommodation; the pupil remained dilated and inactive to light, either direct or consensual, and there was no reflex constriction on accommodation-convergence. In 2 cases the site of the lesion was presumed to be in the brain-stem, although the impact had taken place in the region of the orbit. In 5 others the pupillary disturbance resulted from a paralysis of the oculomotor nerve and was accompanied by the usual manifestations of this lesion. But in 4 cases there were no other signs of nerve involvement and the condition was assumed to be due to a local paralysis of the neuromuscular mechanism responsible for accommodation and pupillary constriction. The term 'internal ophthalmoplegia' has been applied to this condition. In 3 cases the condition was quite transitory, but in the remaining case the dilatation of the pupil and paralysis of accommodation persisted for nine months. The exact nature of this lesion is unknown.

In 2 additional cases blurring of the visual field followed an intra-ocular hæmorrhage, but in both instances the resulting defect in the visual field was the main complaint.

One patient complaining of blurred vision following a car accident was found to have a relative paracentral scotoma and ophthalmoscopic examination revealed a white, atrophic optic disk. This case, therefore, belongs to the group of post-traumatic optic atrophies, which will be discussed later.

Defects in the Visual Field.—These appear to be the commonest complications and the resulting disability is usually a severe one. The nature of this lesion has been subject to a considerable amount of speculation, and it was hoped that a careful analysis of the observations made upon this series of patients would produce some help in the prognosis and management. No great success has been achieved in this direction, largely because it has not been possible to observe a sufficient number of cases in the early stage of injury. Nevertheless, the information so far obtained has allowed some generalizations to be made which may help in patients seen at an early stage.

In 27 patients a defect in the visual field was observed. Evidence that this defect was sustained at the time of injury was obtained in 24 of these patients. In 3 cases the defect was found to be due to an intra-ocular hæmorrhage; in all the remaining 21 patients changes subsequently appeared in the optic disk, indicating that optic atrophy had taken place. In 14 patients the initial loss of vision in the affected eye was complete, while most of the remaining cases showed defects involving more than half of the visual field. The chance finding of a small

visual field defect together with optic atrophy in a patient who had sustained a brachial plexus injury following a car accident, suggests that these minor defects often escape detection by both patient and examiner alike.

In 3 patients the deterioration of vision took place after the first examination. In one, the eye became completely blind during the first twenty-four hours after injury; in the second, blindness occurred some three days after injury following the development of an intra-orbital abscess; while in the third, the eye became blind gradually during the six weeks following the injury.

A review of the clinical findings of these cases shows a remarkable variability in the main features. As these phenomena appear essential for an understanding of the basis of the lesion, they will be dealt with in some detail.

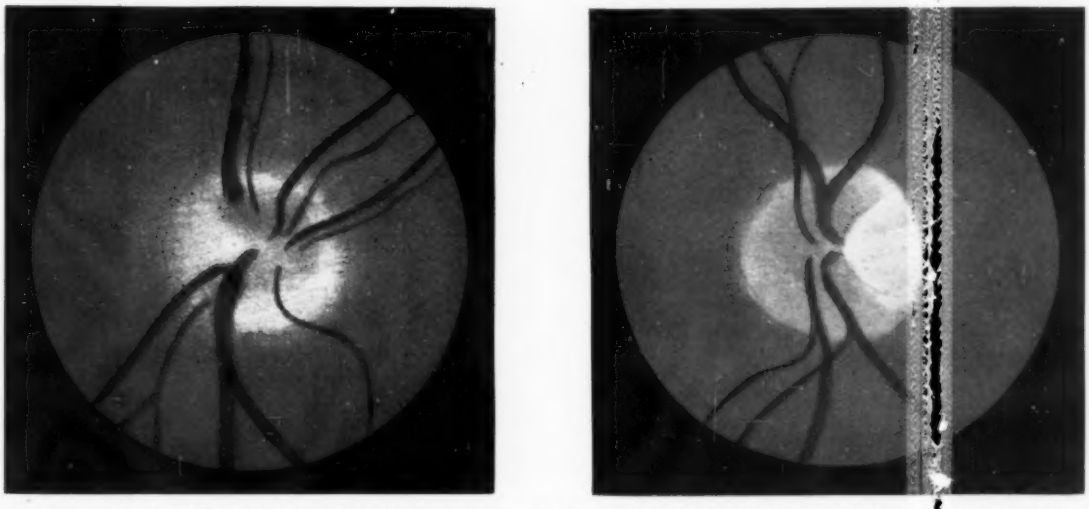


FIG. 185.—*Case* 1. The optic fundi, showing congestion of veins and mild œdema of the optic disk on the affected side.

In all except one patient the initial visual field defect involved at least half of the field of the affected eye. Fourteen of the 20 patients who sustained an immediate unilateral field defect suffered complete loss of vision. In 5 the field defect extended over more than half of the field. In the remaining case a small paracentral scotoma was found at a late stage. This chance finding suggests that many of the minor cases may be missed; on the other hand, during the past five years all patients with a frontal injury have been examined for field defects and no other minor defect has been seen.

The nature of the partial defects is variable; horizontal and vertical hemianopic defects, concentric constrictions, central and paracentral scotomata have all been found.

Thus it appears that the pathological process produced by injury involves the whole or at least the major portion of the nerve, with little gradation between involved and uninvolved portions of the nerve. The lack of any constant pattern tends to exclude the possibility of any constant extraneural cause for the condition.

The Mode of Onset.—The initial features of this lesion cannot always be observed accurately because of the initial unconsciousness, and also because the onset is usually rapid. In three patients some pertinent observations were made concerning the initial stages of the condition.

Case 1.—A man, aged 40, was not rendered unconscious when he was kicked on the side of the forehead in a brawl. He asserted that the loss of vision was so sudden that he thought that the eye had closed. He then became aware that the eye was open and that there was complete loss of vision in this eye.

This instantaneous onset is probably the most common form, but in one patient complete loss of vision was not present until eighteen hours after injury:—

Case 2.—A youth, aged 17 years, when examined four hours after injury, showed a direct reaction to light in the affected eye, and asserted that he could see with this eye. On the following morning the pupil was found to be dilated and lacked a direct reaction to light. There was no perception of light, and the consensual light reflex in the opposite eye was absent.

An unusual onset occurred in one case of delayed bilateral post-traumatic atrophy:—

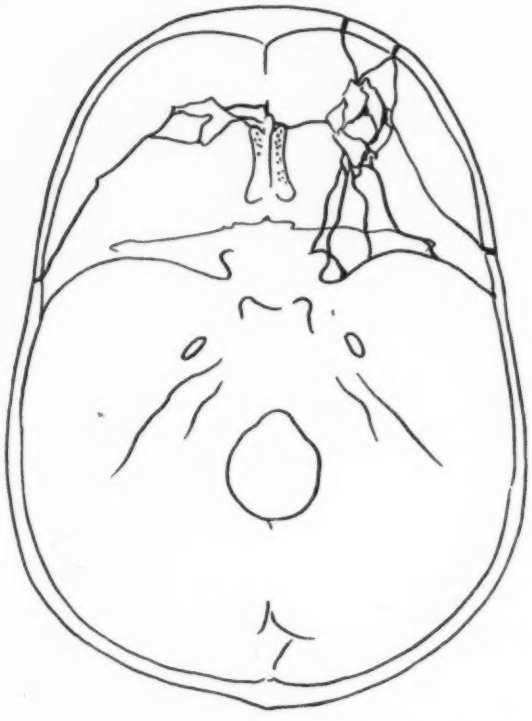
Case 3.—A fireman, aged 37 years, suffered no immediate loss of vision or consciousness after a heavy blow on the outer angle of the left supraorbital ridge. Three weeks later he was aware of some deterioration of vision and eight weeks after the injury no perception of light remained in one eye, while in the other visual acuity was reduced to 6/36 and the visual field was grossly constricted. Examination of the fundi revealed bilateral optic atrophy. Radiographs of the optic foramina revealed no abnormality. The Wassermann reaction was negative.

Although it is agreed that the loss of vision is usually instantaneous, the above cases show that this is not always so. In many cases the early changes are never seen. The observation of a delayed onset suggests that more than one factor operates in the origin of this condition.

Changes observed in the Fundus Oculi.—It has been stated by many authors (Evans, 1929; Rodger, 1943; Turner, 1943) that no early changes are to be observed in the optic disk of those patients

who later develop post-traumatic optic atrophy, and that the normal disk changes gradually to one typical of optic atrophy during the first 2–8 weeks after injury.

In this series, no ophthalmoscopic examination was made during the first two weeks in 14 patients. In 8 of the 10 patients examined in the acute stage of injury changes in the fundus were observed. Although slight in degree, these changes were quite definite and consistent. Distension of veins, blurring of disk margins, and a general pinkish coloration of



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the disk as compared with the normal unaffected eye, were the changes observed in 4 patients in whom they persisted for 3–7 days and then receded; the disk changing to the typical atrophic state in 3–5 weeks. *Case* 1 quoted above showed these changes when observed on the second day after injury (*Fig.* 185). In other cases seen in the acute stage of injury no abnormality was found on ophthalmoscopic examination.

Two additional patients merit attention in this respect: The first, seen twenty-four hours after injury, had slight distension of veins in the affected blind eye, but the disk had a white, atrophic appearance at this stage and was not greatly changed five weeks later; the second patient was a child who showed a white, swollen disk when seen 3–4 weeks after injury.

Thus it would appear reasonable to assume that in many cases changes may be observed in the early stages. This opinion is supported by other observers (Blakeslee, 1929; Goulden, 1929). The congestive changes may result from venous obstruction or from congestion associated with a vascular reaction to injury. It has been suggested that in partial lesions (Goulden, 1929) they may be the result of an extension of subarachnoid or subdural hæmorrhage along the optic nerve-sheath. The changes are not themselves the cause of the subsequent atrophic change, but are phenomena accompanying the initial lesion.

Recovery.—Recovery from the initial defect is usually incomplete. When the initial defect was extensive there was usually considerable delay in recovery, and this was usually inadequate. Recovery of useful vision was not common after initially complete defects. Of the 14 patients who had a complete unilateral loss of vision in the acute stage only 3 recovered any vision. In 1 patient, some recovery was present ten days after injury; in another it appeared three weeks later, while in the third it did not appear until five months after injury. The amount of recovery was greatest in the first patient, and when once established it appeared to advance

B

FIG. 186.—*Case* 4. A, Drawing showing the fractures of the anterior fossa of the skull. B, A sketch of the contused laceration of the optic nerve adjacent to the fractured anterior clinoid process.

rapidly for 2–3 weeks and then slowed down. Useful vision was obtained in 2 patients who had sustained an initial complete loss of vision and in 3 patients in whom the initial defect was incomplete.

No evidence of recovery was observed before the tenth day. These findings appear intermediate between those of Turner (1943), who states that useful vision will only be obtained if recovery commences by the third or fourth day, and Hughes (1945), who asserts that recovery in the first four weeks is minimal.

Changes observed in the Optic Nerve and Optic Chiasm.—In 6 cases in the series the opportunity arose to inspect the affected optic nerve or chiasm. As these observations present important information concerning the mechanism of the injury, they will be described in detail. One case was observed at autopsy:—

Case 4.—This patient was involved in a head-on car accident. When admitted to hospital soon after the injury he was in deep coma. The right eye was proptosed and the pupil did not react directly to light, nor was the consensual light reflex elicited. Death occurred thirty-six hours after injury. At post-mortem examination comminuted fractures of the right orbital plate were found. One fracture line passed through the roof of the optic canal, while another severed the anterior clinoid process (*Fig.* 186). There was a laceration of the optic nerve opposite the anterior clinoid process and a contusion extended from the chiasm along the nerve to the apex of the orbit.

A similar finding was found in another patient during an operation to close a communication

between the posterior ethmoidal air-cells and the subarachnoid space :—

Case 5.—A truck driver sustained a crush injury of the skull. Both eyes were rendered blind instantaneously. When first seen six days after the injury, he had developed a meningitis. The right fundus appeared to be within normal limits, but the left was obscured by a large intraocular hæmorrhage. Radiographs showed a fracture involving the frontal sinus and ethmoidal air-cells, and air was present in both lateral ventricles. At operation a large bone defect was found behind the cribriform plate through which air bubbled into the cranial cavity (*Fig.* 187). The left optic nerve appeared normal, but the right was grossly contused at the level of the clinoid process and this structure was found to be shorn across

A

and retained in position by a few dural tags. Other fracture lines passed across the lesser wing of the sphenoid on the right side. Later, useful vision followed the slow resolution of the hæmorrhage in the left globe, while the right remained blind. Both optic disks eventually showed pallor and atrophy.

The changes in the third case were of considerable interest in relation to the development of the visual defect and its subsequent recovery :—

Case 2.—The patient became completely blind in the affected eye during the first eighteen hours after injury. Ophthalmoscopic examination showed some distension of veins in this eye as compared with the normal eye. Radiographs showed a fissure fracture extending from the parietal region to the lateral wall of the orbit. No fracture was seen in the region of the optic foramen. Because of the evidence of a progressive lesion, the orbit, optic nerve, and optic chiasm were explored by the transfrontal route. A small hæmatoma (4–5 ml.) was found on the lateral wall of the orbit. The optic chiasm and the intracranial portion of the optic nerve appeared normal. The roof of the optic canal was removed and the dural sheath opened up throughout the length of the canal and for a short distance into the orbit. No extravasation of blood was found within the sheath. The nerve was seen to bulge up through the incision and the space made by the removal of the roof of the canal. No immediate improvement occurred, but at the end of a week light perception appeared in the upper portion of the field and this area expanded until the defect was limited to a horizontal hemianopia.

The influence of operation in this case is largely speculative until some more definite hypothesis of the pathology of the condition can be applied.

The fourth case was less conclusive because operation was requested at a late stage in the evolution of the lesion, but the findings are still of interest :—

Case 6.—A motor-cyclist sustained a head-on injury, in which he was rendered blind in the right eye and also had a complete third-nerve palsy on this side. When seen five weeks after injury, the right optic disk was atrophic and the visual field on the left side showed a partial defect in the left upper nasal quadrant. At exploration the chiasm and right optic nerve were covered with thin filmy adhesions. On separating these the right optic nerve was found to be thin, grey, and atrophic (*Fig.* 188). The roof of the orbit and optic canal was removed. No gross lesion was found within the canal,

B

FIG. 187.—*Case* 5. Operation sketch (A) showing the dural tears overlying the fractures in the anterior fossa and the lacerated optic nerve lying beside the fractured anterior clinoid process (diagrammatically represented in (B)).

but a fracture was found passing along the lateral aspect of the canal, i.e., between the canal and the superior orbital fissure. The patient asserted that he could perceive light for a few days after the operation but then the eye remained blind. An interesting feature of this case was the rapid recovery of the third-nerve palsy which began immediately after the operation.

One case of chiasmal injury was explored eighteen months after the original injury because there had been a recent reduction in visual acuity, and a chiasmal arachnoiditis was suspected to have developed around the chiasmal injury which had produced a bitemporal hemianopia at the time of the injury.

Case 7.—The patient, aged 40, showed radiographic evidence of old fractures of the left frontal bone and roof of the left orbit. Visual field examination showed a bitemporal hemianopia with poor visual acuity (V.A.R. 6/9, V.A.L. 6/60). After an encephalogram had demonstrated defects in the filling of the cisterna chiasmaticus, this region was explored (*Fig.* 189). Both optic nerves were shrouded with dense scar, which could be easily removed from the left optic nerve and chiasm. The nerve appeared normal but the chiasm was yellow and shrunken. The operation was followed by an improvement in the acuity in the left eye (6/36) with some enlargement of the temporal half of the visual field.

The last case developed a bilateral loss of vision some weeks after injury and the initial features have already been described :—

Case 3.—This patient, when first seen, was completely blind in the left eye, while the visual acuity in the right was reduced to 3/24. After an encephalogram had again suggested a chiasmal arachnoiditis, exploration of the chiasm was carried out, but only a few filamentous adhesions were found. The optic nerves and chiasm appeared normal.

From these direct observations it will be readily appreciated that there is no single constant aetiological factor in the production of the lesion.

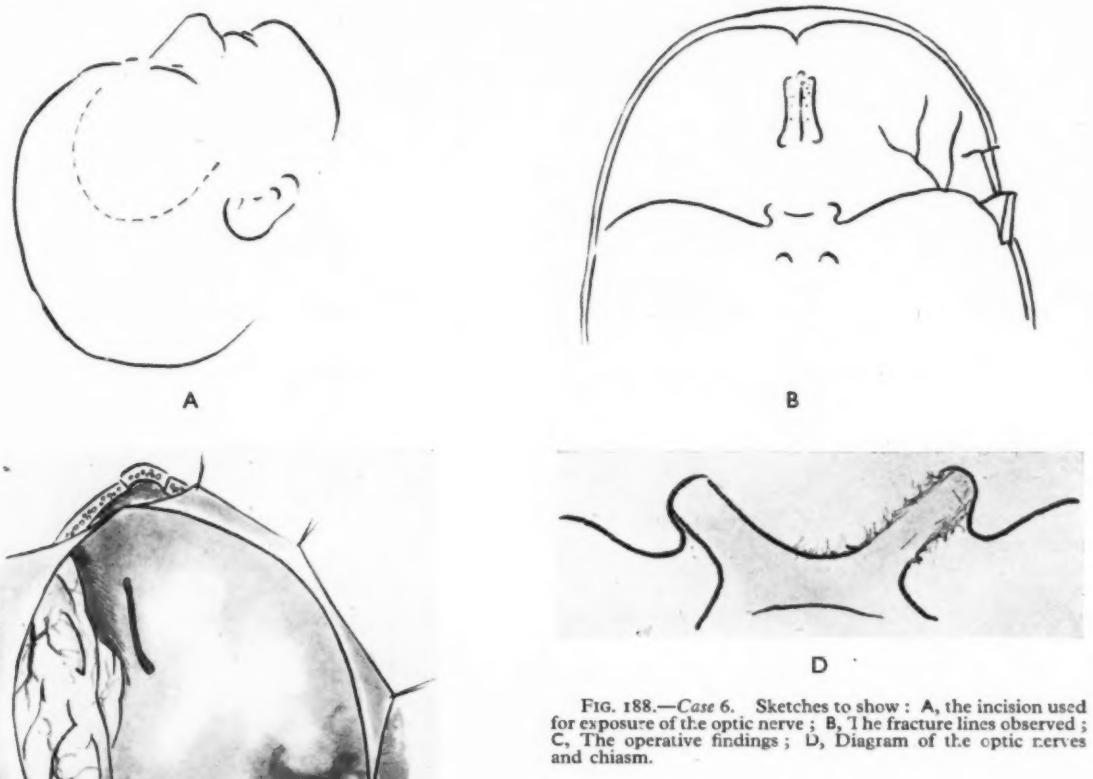


FIG. 188.—*Case* 6. Sketches to show: A, the incision used for exposure of the optic nerve; B, The fracture lines observed; C, The operative findings; D, Diagram of the optic nerves and chiasm.

Radiological Evidence of Fracture.—No fracture of the optic foramen or anterior clinoid process was demonstrated in the radiographs in any of the cases in the series; in the 2 cases in which the lesion was demonstrated at operation no fracture was seen in subsequent radiographs.

In 14 patients radiographs showed fractures involving the walls of the orbit. In only one of these did it seem likely that the intra-orbital portion of the nerve was involved. In the radiograph, a fragment of the roof was seen to be depressed and rotated so that it lay at right angles across the course of the nerve.

No fracture of the skull was demonstrated in 3 patients.

The difficulties encountered in the radiographic demonstration of a fracture through the optic canal or anterior clinoid process are well recognized. Turner (1943) considers that the radiological demonstration of a fracture of the optic canal is a rare event, but Rowbotham (1949) found fractures involving the canal in 3 of the 7 cases described, while Rodger (1943) was able to demonstrate a distorted foramen on the affected side in 9 out of 13 cases. The radiographic demonstration of a fracture depends upon the disposition of the fracture and on technical factors associated with the positioning of the patient and the quality of the X-ray apparatus; these are variable factors and it would seem preferable to determine how frequently these fractures occur and what significance may be attached to them. A comparison of the various series published over a number of years shows considerable disparity. Some authors draw largely upon the experience of von Hoelder, who found 54 fractures of the optic canal in 88 fractures of the base of the skull, but in a critical examination of this material (Pringle, 1922) it is pointed out that 42 of these cases were gunshot wounds of an unusual type. The frequency of a fracture of the optic canal as a cause of this condition is also discussed by Rawling (1904), who suggests that a fracture of the anterior clinoid process is more probable.

Possibly the most useful figures are set out by Pringle (1922), who describes 395 cases of skull fracture. Of the 186 survivors only one suffered loss of vision. Among the 209 fatalities, 174 were subjected to post-mortem examination. Only 13 showed a fracture involving the optic foramen, and in none

of these was there evidence of injury to the nerve itself. The anterior clinoid process was fractured

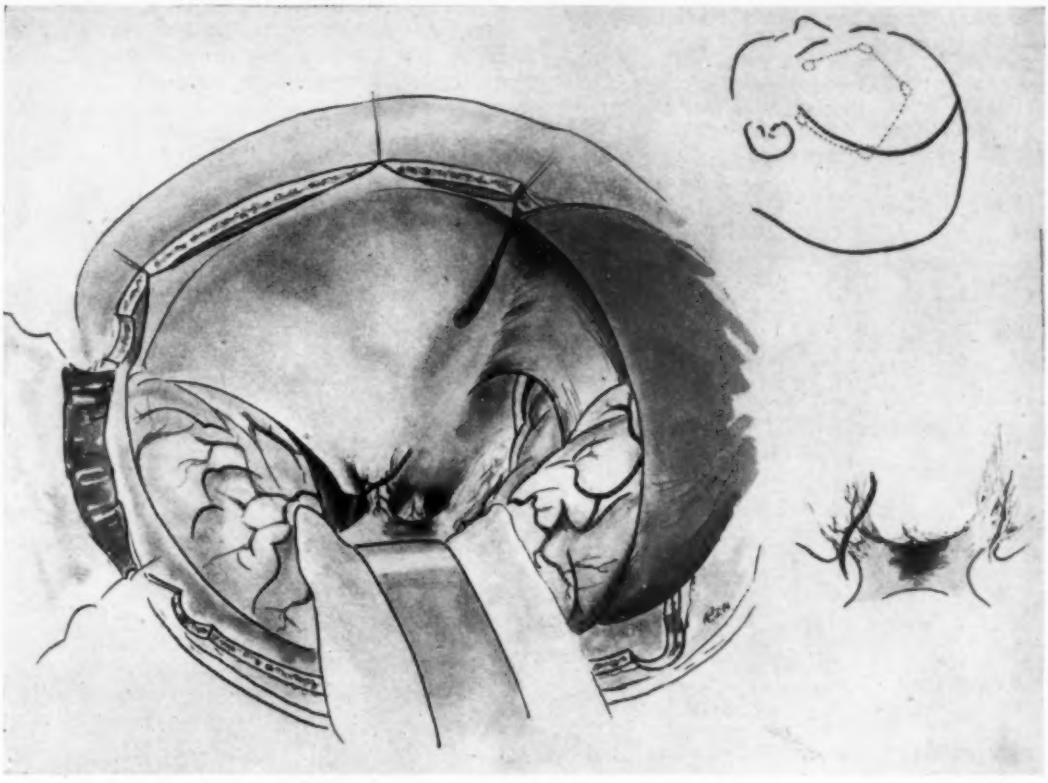


FIG. 189.—*Case* 7. Operation sketch showing dense, vascular arachnoidal adhesions surrounding the optic nerves and the pigmented, atrophic central portion of the optic chiasm.

in 6 cases, but again no gross lesion was found in association with the fracture.

Summing up this evidence one may say that fracture of the optic canal or clinoid process occurs in about 10 per cent of fatal head injuries. It is reasonable to assume that in non-fatal head injuries the incidence is lower. When present it is frequently, but not necessarily, associated with a lesion of the nerve. When a lesion is present the possibilities of radiographic demonstration of a fracture are limited.

Effects of Secondary Lesions.—Deterioration of vision may follow some secondary effect of the orbital injury. In this series the most striking example followed the development of an intra-orbital abscess. A search of the literature revealed one similar case, described by Hilton (1950).

FIG. 190.—*Case* 8. Showing the chemosis, proptosis, and fixation of the globes due to bilateral intra-orbital abscesses. A trickle of cerebrospinal fluid is seen at the left nostril.

Case 8.—This patient, involved in a head-on car accident, struck his face and forehead on the back of the seat in front of him. Swelling developed early and a radiograph showed fractures of the bony block between the orbits. On the third day the vision in the right eye became impaired, and two days later the eye was completely blind. When examined on the sixth day, there was extreme orbital swelling and chemosis (*Fig.* 190).

Gross bilateral proptosis was present and the globes were immobile. Vision in the left eye was blurred, but the pupil reacted briskly to light. No perception of light was present in the right eye and no direct reaction to light was present. Examination of the fundi showed no abnormality on the left, but on the right the fundus appeared pushed forwards, it was pallid, and the vessels were exsanguinated. Cerebrospinal fluid leaked from the nostrils and marked neck stiffness was present. An immediate operation was performed and the floor of the anterior fossa exposed by the transfrontal route. Pus was found in both frontal sinuses and in all the ethmoidal air-cells. Both orbits were unroofed and a large abscess cavity was found in each orbit (*Fig.* 191). After evacuation of these cavities, penicillin tubes were introduced into each and brought out in the temporal region. The infection rapidly subsided and the proptosis diminished. About ten days after operation the right fundus became revascularized but the eye remained blind.

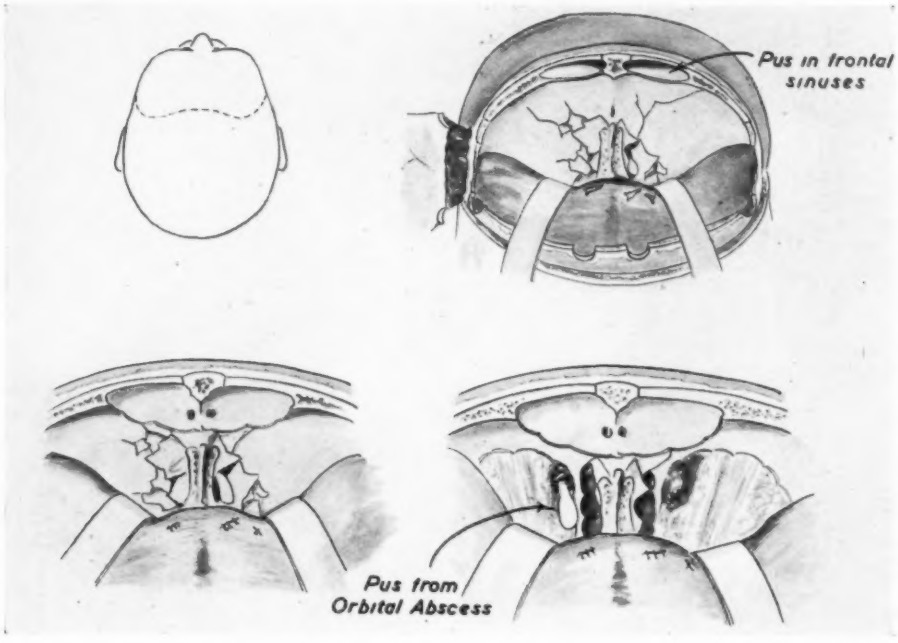


FIG. 191.—*Case* 8. Operation sketch showing, top left, a fluid level of pus in the frontal sinuses, fractures of the central portion of the anterior fossa with pus leaking from ethmoidal cells. Three dural tears are seen. On the right, the posterior walls of the frontal sinuses have been removed, while below, the orbital roofs have been removed and pus exudes from the intra-orbital abscess on the left side.

In one other patient (*Case* 7) delayed deterioration of vision was due to the development of arachnoiditis in the region of the optic foramina. This complication has been described by numerous authors. Other factors responsible for involvement of the nerve have been described. Lillie and Adson (1934) describe 2 cases in which the optic nerve was involved some time after injury by the development of callus at the fracture site. It is interesting to note that in one case the initial injury which caused the fracture did not produce a demonstrable lesion of the nerve. Pringle (1922) found blood under tension in the optic nerve-sheath in 3 patients explored at least two months after injury.

DISCUSSION

A survey of this material reveals a number of observations from which it is possible to form a working hypothesis which may assist in prognosis and management of these injuries. It must be remembered that the optic nerve differs from other cranial and spinal nerves in that its nerve-fibres have no neurilemma sheath, and they are invested in a layer of pia arachnoid and dura mater until they reach the globe. In the relatively short course of 4 cm. the nerve passes through the orbital fat, the optic canal, and the subarachnoid space of the cisterna chiasmaticus. A defect in the visual field will occur when conduction is arrested at any point along this pathway. Other relevant phenomena, such as changes in the optic disk and radiographic abnormality, will depend largely upon the site of the injury.

To insist that all such lesions are due to fracture of the anterior clinoid process is absurd. It is equally absurd to associate all lesions with a fracture of the canal. Perhaps it is simpler to regard the optic nerve as analogous to the spinal cord, and that conduction may be arrested in a manner similar to spinal injuries. Thus the lesions may be due to concussion, contusion, laceration, compression, and vascular obstruction of the nerve at any point along its course. The lesions may be produced by :—

1. Structural derangement of the orbital contents at the moment of impact. This may be of very short duration.
2. Actual involvement of the intra-orbital portion of the nerve by fragments of the orbital roof.
3. Fracture of the optic canal.
4. Fracture of the anterior clinoid process.
5. Traction or torsion of the optic nerve by displacement of the brain away from or towards the side of the lesion during a mass movement of the brain within the skull immediately following the impact.

Laceration of the nerve is followed by permanent loss of function of the portion involved, together with varying degrees of permanent derangement of adjacent portions. Within the nerve itself, swelling and hæmorrhage tend to spread up and down along the line of the fibres, with little lateral spread. This process tends to cause pressure on individual nerve-fibres more or less equally, thus suggesting a reasonable explanation for the frequent 'all or nothing' effect on the visual field.

The effect of vascular occlusion may also be added to the hæmorrhage and œdema, and the partial or patchy occlusion of vessels may explain the bizarre forms of visual defect sometimes encountered.

The situation of the lesion with respect to the optic disk and the local derangement of vessels probably accounts for the variation seen in the optic disks and the period which elapses before primary optic atrophy is apparent. In addition, the presence of blood in the subarachnoid or subdural spaces around the optic nerve may also influence the appearance of the optic disk in the acute stage of injury. In the presence of such lesions one would expect to find all gradations between normality and moderate congestion.

Finally, it must be recognized that post-traumatic optic atrophy may follow a number of secondary effects.

Management.—Considerable divergence of opinion exists upon this subject. Lyle (1945) suggests that operation should be carried out as soon as the patient's condition permits, while Stallard (1946) asserts that operation is rarely of value. This variation of opinion would appear to be due to the fact that it has not been possible to formulate indications for operation from the meagre information available concerning the nature of the lesion. In addition, it is still difficult to obtain a proper evaluation of the results of operation.

Comparison with spinal cord injuries probably gives the best guide for the management of these cases. Operation in immediate complete lesions is usually unprofitable, but in those patients with progressive deterioration of function after injury, operation will usually disclose a definite cause. If it is possible to relieve this, a reasonable degree of return of function is possible. On present evidence, it is considered reasonable and, indeed, essential to explore all those patients showing progressive deterioration of vision. The transfrontal route is employed so that the optic chiasm and the intracranial portion of the optic nerve may be inspected. The roof of the optic canal and the adjacent portion of the orbital roof are removed and the dural sheath of the nerve opened to expose all the vulnerable portions of the nerve.

DOUBLE VISION

Double vision is a relatively common sequel of the frontal head injury. In many cases the disability disappears during the subacute stage of injury, but in others mild degrees of double vision delay resumption of activity. In a few, the defect is of a permanent nature and constitutes a very considerable disability.

In this series of cases, a paresis of an extrinsic muscle or displacement of the globe were obvious causes of the diplopia, the cases being distributed as follows :—

Paresis of an extrinsic muscle	15 patients
Displacement of the globe	5 patients

Paralysis of an extrinsic muscle, without diplopia, occurred in 4 patients, there being a gross field defect in the affected eye as a result of involvement of the optic nerve. In this total of 19 patients the distribution of the nerve involvement was as follows :—

Oculomotor nerve	12
Trochlear	2
Abducens	12

Goulden (1929) states that the oculomotor nerve is rarely involved and that it is the abducens which suffers most frequently. The figures given above correspond closely with those quoted by Turner (1943) who, in a series of 1550 head injuries, found that the oculomotor and abducens nerves were equally involved.

Some interesting findings were obtained when the direction of the blow and the site of the impact were studied. With one exception, in all the patients in whom the oculomotor nerve was involved alone, the impact occurred in the central frontal region, while in the lesions of the abducens nerve the impact was on the lateral side of the forehead or orbit in 5 patients, in 1 the head was squeezed from side to side ; in only 1 case the impact occurred in the central frontal region.

The oculomotor nerve lesion is usually in the orbit or the sphenoidal fissure ; in 7 patients multiple fractures of the orbit were present. In 2 cases the effect of operation upon the nerve lesion was remarkable. In neither case was the operation designed for the relief of diplopia. In the first a decompression of the orbit and optic canal was carried out in an optic nerve exploration, while in the second a depressed orbital roof was elevated. In both cases recovery of oculomotor nerve function commenced within a day or two of operation and was complete in 2–3 weeks—a more rapid recovery than is usually seen. No satisfactory explanation of a specific nature could be made in either instance.

In 2 cases of oculomotor nerve involvement, localization within the brain-stem appears to be the only satisfactory explanation of the syndrome produced. In the first of these patients there was a gradual development of a third-nerve palsy on the left side following a fall on the face. At the same time there was right-sided ataxia and weakness. The condition reached its full development in forty-eight hours and persisted for several weeks. The full significance of this case was not appreciated at the time and examinations were inadequate. In the second patient the onset was similar :—

Case 9.—While out riding, this patient was thrown forward upon the head of the horse, striking the region of the left orbit on a bony prominence of the horse's head. Thirty minutes later the left eye would not open and a small ecchymosis of the eyelid had appeared. At the end of twenty-four hours he had a complete internal and external ophthalmoplegia (*Fig.* 1-2). There was a right lower facial weakness, and ataxia of the right hand and leg. A dysarthria of the cerebellar type was also present. Improvement occurred in the ophthalmoplegia, but the

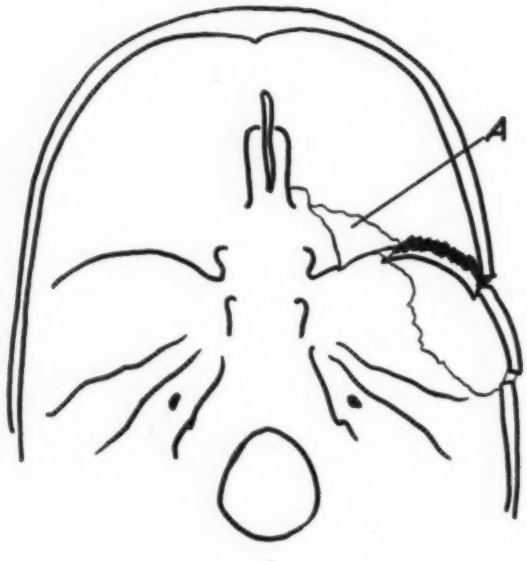
ataxia and slurring of speech are still present nine months after the injury (*Fig.* 193).

This lesion is of interest in relation to the mid-brain injuries recently described by Kremer, Russell,

FIG. 192.—*Case* 9. The complete internal and external ophthalmoplegia following a blow on the orbital region.

and Smythe (1947), but the mechanism is still obscure.

On only one other occasion did an internal and external ophthalmoplegia occur :—



A

Case 10.—This patient sustained a severe impact in the right fronto-temporal region and a complete internal and external ophthalmoplegia was found immediately after the injury. This persisted until the patient died of an intercurrent infection eight days later. At autopsy depressed fractures of the greater and lesser wings of the sphenoid were found. The lateral margin of the superior orbital fissure was displaced medially, compressing the nerves as they entered the orbit (*Fig.* 194).

In those cases with abducens palsy, the site of the lesion is not easily demonstrable by radiographic examination, but there is little doubt that in those cases where nerve or muscle are not involved in the orbit the commonest site is in the region of the apex of the petrous portion of the temporal bone. This was suggested by Wolff (1928), and has been demonstrated experimentally in crushing injuries by Russell and Schiller (1949). Gurdjian, Webster, and Lissner (1950) have demonstrated that this transverse fracture across the apex of the petrous follows impacts directed towards the lateral aspect of the skull.

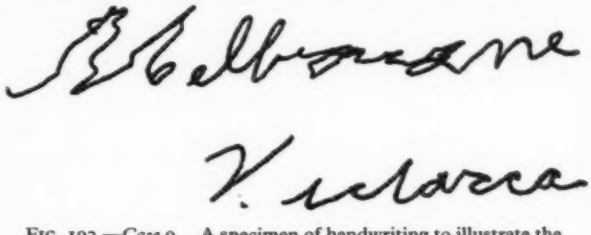
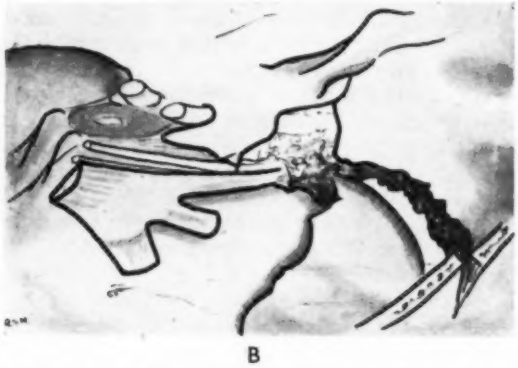


FIG. 193.—*Case* 9. A specimen of handwriting to illustrate the accompanying ataxia of movement.

Thus it seems that the main factor in the production of the disturbance in the oculomotor mechanism is the direction and situation of the impact, the oculomotor nerve being more prone to damage in direct frontal blows, while the abducens is involved in those disposed more laterally.

When diplopia is due to displacement of the globe, two factors may be present : the displacement may be beyond the limits within which muscle activity may compensate, or there may be a concomitant disturbance of muscle function by displacement of the walls of the orbit. The exact proportion of each factor operating is usually impossible to determine, but in 6 patients there was gross displacement of the orbital margins, while in only 2 patients the depression of bone fragments occurred behind



B

FIG. 194.—*Case* 10. A, Sketch showing the situation of the depressed fragments ; and B, Sketch after removal of the depressed fragment A, showing the third, fourth, and ophthalmic nerves entering the orbit.

the orbital margin (*Figs.* 195, 196). An abscess on the medial wall of the orbit caused diplopia in 1 patient, while in 2 others the protrusion of brain substance into the orbit was the initial cause.

Diplopia due to simple fractures of the orbital floor—zygomatico-maxillary fractures—is common, but isolated injuries of this nature are not included in this series, the majority of these fractures being

corrected in the Department of Facio-maxillary Surgery.

Management.—Many cases with initial diplopia following injury fuse images later and there is no

FIG. 195.—Photograph showing the medial and downward displacement of the globe resulting from a blow in the lateral aspect of the orbit.

persistent disability. But displacement of the globe in the presence of deformity of the orbit usually necessitates operative reduction. The earlier this is carried out the more complete the reduction. Early reposition of fractures of the zygoma is important and co-operation with the facio-maxillary surgeon must not be delayed. Depressed fractures of the upper margin of the orbit present no difficulty if the fragments are not comminuted. When the depression is laterally placed an incision just behind the hair line gives sufficient access, but for extensive fractures of the supra-orbital margin and related portions of the frontal bone and frontal sinus, a frontal scalp flap gives a better exposure and an osteoplastic bone-flap may also be necessary.

The reduction of the deformity is easy in most cases, but occasionally the bone around the orbit is so comminuted that careful reposition and wiring of fragments fails to raise the depressed supra-orbital ridge, as in the following case :—

Case 11.—A motor-cyclist, in a head-on collision, sustained comminuted fractures around the left orbit together with fractures of the long bones. There was depression of the globe with diplopia. The development of an intracranial pneumocele necessitated a fascial graft in the frontal sinus region. When this had been placed

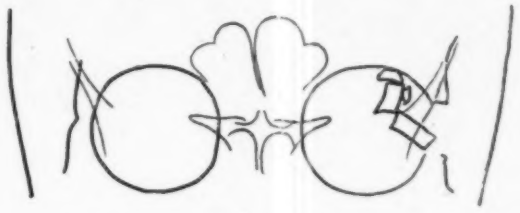


FIG. 196.—Tracings of the radiographs of the same patient, showing the depressed fragments of the lateral wall of the orbit.

in position the fragments were carefully wired in position (*Fig.* 197). Although the diplopia was corrected, the wired fragments dropped back and the resulting cosmetic effect was poor.

The procedure adopted in another case which was referred some time after injury suggests that it may be better in some extensively comminuted fractures to remove all the fragments during the initial débridement. The fragments are then retained and an accurate mould of the defect is made from them so that an acrylic resin plate can be made. This is inserted at a second operation.

Case 12.—In a head-on accident, the patient sustained a compound comminuted fracture of the right frontal region. During the initial débridement all the loose fragments of bone had been removed and, when first examined three months later, the patient was found to have a post-traumatic optic atrophy of the left optic nerve and a gross deformity of the right orbit due to the loss of the entire supra-orbital margin with a considerable portion of the adjacent bone. A mould of dental compound was prepared and from this an acrylic plate was made and inserted without difficulty in a single operation (*Figs.* 193–200).

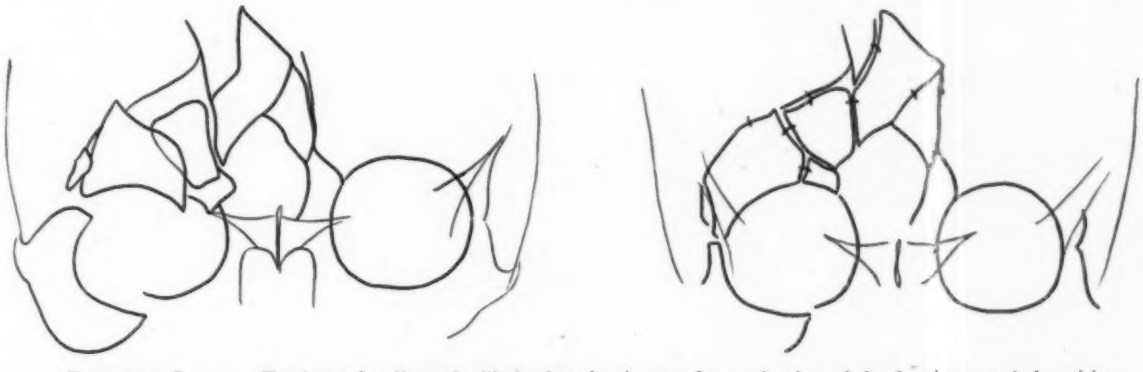


FIG. 197.—*Case* 11. Tracings of radiographs illustrating the degree of comminution of the fragments and the wiring necessary to retain their position.

The cosmetic result of this simple procedure was very satisfactory and the comparison with the

previous case suggests that the initial removal of comminuted fragments, followed by a plastic procedure for the resulting defect, may present less difficulty and achieve a better cosmetic result.

DISPLACEMENT OF THE GLOBE

Displacement of the globe from its normal situation in the orbit is a common sequel to a frontal injury. Some protrusion of the globe is usual with the development of the orbital hæmatoma, but is usually masked by the associated swelling and bruising of the loose areolar tissue of the lids. In 16 patients in this series serious displacement of the globe occurred. The type and frequency of the displacement is indicated by the following :—

FIG. 198.—*Case* 12. The patient seen after the inital débridement of the compound frontal fracture.

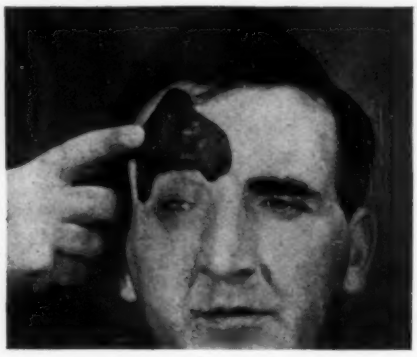


FIG. 199.—The mould from which the acrylic resin plate is made.

FIG. 200.—The patient after the introduction of the acrylic resin plate.

FIG. 201.—*Case* 13. The marked proptosis associated with infection of the orbit from the maxillary antrum.

	Cases
Protrusion or proptosis	10
Downward displacement	3
Medial displacement	2
Posterior displacement	3

In one patient the proptosis was due to distortion of the walls of the orbit to such a degree that removal of the globe was necessary.

Orbital infection caused proptosis in 4 patients. In two of these there was evidence that the infection originated in the paranasal sinuses. A very severe degree of proptosis occurred in the first case.

Case 13.—A boy of 15 years sustained a fracture of the orbit, which also involved the infected antrum below it. During the five days which followed the injury a gross degree of proptosis developed (*Fig.* 201), which subsided after the reduction of a displaced zygoma and adequate administration of antibiotic drugs.

In the second of these cases, abscess formation occurred in both orbits following fractures of the anterior fossa in the presence of an acute sinus infection. Both globes were proptosed and fixed, and occlusion of the retinal vessels of one eye resulted in permanent blindness. The course of this patient has been described. A similar but smaller abscess occurred on the medial wall of the orbit in another patient some days after the repair of a compound fracture of the opposite fronto-temporal region. No direct relationship was established between the two lesions.

Protrusion of brain substance into the orbit caused proptosis in 2 patients. In the first a pulsating exophthalmos was present, suggesting the presence of a traumatic carotico-cavernous aneurysm :—

Case 14.—A 15-year-old boy struck his head on a projection from the dashboard of a car which had become involved in a head-on collision. He sustained lacerations around the lids but no damage to the globe. Early radiographs failed to show a fracture of the skull. During the next three days the swelling of the upper lid increased, the globe protruded, and pulsation became apparent (*Fig.* 202). This could be increased by jugular compression. Further radiographs of the skull showed that a large, thin flake of bone from the orbital roof had been driven upwards into the anterior cranial fossa. When the orbit was approached by the transfrontal route the flake of bone was found presenting under the upper surface of the frontal lobe. There was gross laceration of the orbital surface of the frontal lobe overlying a large defect in the dura and the roof of the orbit. Pulped brain had prolapsed into the orbit in considerable quantity (*Fig.* 203). After a repair of the region the pulsation ceased and the orbit soon resumed a normal appearance.

It is noteworthy that in all those patients who presented with gross protrusion of the eye following trauma, a correct assessment of the lesion could not be made before operation. As the risks attendant upon some of these conditions were very great, an immediate inquiry into the cause of the condition had to be made and the necessary treatment undertaken without delay.

Retraction of the globe into the orbit usually results from depressed fractures of the orbital

margins or atrophy of orbital fat due to infection, but in one case in the series the apparent retraction of the globe was due to a lengthening of the long axis of the orbit following a crush injury along the transverse axis of the skull.

FIG. 202.—*Case* 14. The pulsating proptosis before and after a dural repair of a defect in the orbital roof.

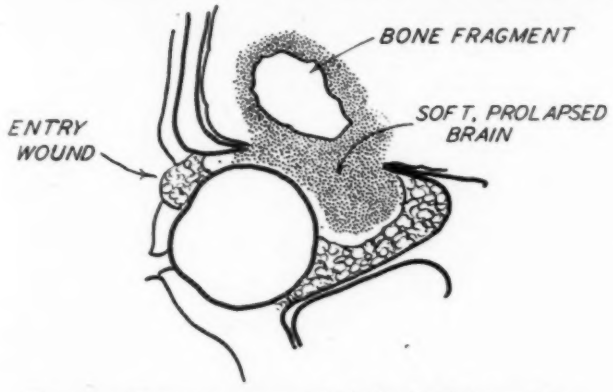


FIG. 203.—Diagram to represent the lesion found at operation in *Case* 14.

Case 15.—A child of 4½ years fell beneath a horse-drawn vehicle and a wheel passed over the child's head. Some fifteen minutes later the child was admitted to hospital in a conscious but severely shocked condition. The skull was obviously distorted; the left side of the head being flattened. A wide fissure fracture could be felt running across the frontal region. Brain substance exuded from the nostrils. A large depression in the parietal region was elevated on the same day, but subsequently the child developed a severe pneumococcal meningitis and further operative repair was delayed. When the meningeal infection was controlled the facial swelling had subsided and it was possible to ascertain that there was no visual impairment, in spite of the gross orbital deformity. Radiographs now showed that almost the entire frontal bone had been displaced forwards and laterally, leaving a wide gap across the roofs of both orbits so that the globes now appeared retracted behind the orbital margins. The left external angular process was displaced to the right so that it could be palpated beneath the left eyelid and limited the field of vision in this eye. A fascial repair of the anterior fossa was carried out, but the interposition of soft tissues through the wide gap in the orbital roofs, together with the necessary delay in operating, did not allow adequate reduction of the deformity. (*Fig.* 205.)

In two patients some abnormality of the sympathetic denervation appeared to be the cause of the orbital disturbance. One patient sustained a fracture across the base of the skull, with a right facial paralysis and a right Horner's syndrome with enophthalmos (*Fig.* 206). The second patient sustained a right parietal fracture with a large extradural

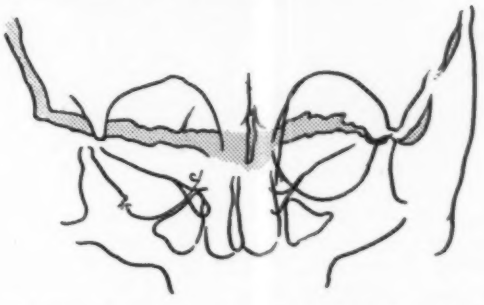


FIG. 204.—A tracing of a postero-anterior radiograph of the skull showing the displacement of the frontal bone to the left and the wide fissure (shaded) which passes across the anterior fossa of the skull.

hæmorrhage from the right middle meningeal vessels. During the two weeks after the evacuation of the clot he developed an exophthalmos of the left

FIG. 205.—*Case* 15. A photograph showing the prominence of the right external angular process which now overlies the right globe and limits lateral vision.

eye (*Fig.* 207). No other aetiological factor could be ascertained.

In the remaining cases lateral and downward displacements of the globe were due to depression

of the orbital margin. Radiographs usually give adequate information concerning the cause of the displacement and their management presents no difficulty.

These examples indicate that displacement of the globe is of considerable importance. There must be no delay in assessing the nature of the displacement nor in arranging for early reduction of the deformity in order to prevent late complications and to diminish the cosmetic defect of the injury.

FIG. 206.—A Horner's syndrome and facial paralysis associated with a fracture of the base of the skull.

SUMMARY

Derangements of the orbit affecting visual function are a frequent sequel of the head-on injury and often present as a complication of a head injury which may be variable in severity.

The common complications affecting the visual mechanism are: deterioration of vision, double vision, and displacement of the globe.

Deterioration of vision may be due to defective accommodation or defects in the visual field subsequent to injuries to the globe or the optic pathway.

The problem of post-traumatic optic atrophy is discussed in the light of the data obtained from 21 patients, particularly in respect to the following: (1) the visual field defect; (2) the mode of onset; (3) the changes observed in the fundus oculi; (4) recovery; (5) the changes observed in the optic nerve and chiasm at operation and at autopsy; (6) radiological evidence of fracture; (7) the effects of secondary lesions in the orbit.

The aetiology and pathology of 'post-traumatic optic atrophy' is discussed and the management of the condition is considered.

The causes of diplopia are classified and the frequency of nerve involvement discussed in relation to the nature of the impact and the situation of the lesion. Diplopia due to displacement of the globe is discussed in relation to management.

Finally, displacements of the globe itself are subject to analysis and examination in relation to diagnosis and management.

I am indebted to the members of the staffs of the Royal Melbourne Hospital and the Children's Hospital, Melbourne, for referring many of the patients whose injuries are recorded in this paper, and also for their help and advice in the management of related problems. To colleagues in country centres who have referred other cases in the series, I would also express my gratitude. Due acknowledgement is made to Miss M. Turnbull for her secretarial assistance.

FIG. 207.—Unilateral exophthalmos following a head injury in which an extradural hæmorrhage occurred on the opposite side.

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DIVERTICULOSIS OF THE JEJUNUM—A CLINICAL ENTITY

By I. M. ORR and J. Y. W. RUSSELL, Preston

The occasional observation at operation or autopsy of single or multiple diverticula of the jejunum usually evokes little comment or surgical curiosity. Such diverticula are sometimes reported by the radiologist as an afterthought, as though they were of no pathological significance, and a deliberate investigation of the small intestine to demonstrate such variations from normal, as a possible explanation of dyspepsia or hæmatemesis, is seldom made. The experience of the writers, however, is that such diverticula are met fairly frequently, and that they may cause symptoms of chronic dyspepsia or give rise to complications of such magnitude that they should be considered in the differential diagnosis of indigestion and as a possible cause of hæmatemesis or acute abdomen.

Many writers have reported single cases and a few have discussed the subject from a wider angle. This paper will describe a short series of patients seen by the authors and will review some clinical and pathological aspects of the subject.

History.—Fischer (1900) mentions reports by Sommervit in 1794 and Voigtel in 1804 describing the condition, and Klebs (1869) described the relationship of the artery to the sac. The earliest records in English medical literature appear to have been made by Cooper (1804) and Osler (1881). These reports were of findings at autopsy. The earliest description of a living case was made by Gordinier and Sampson (1906). Helvestine (1923) observed the constant appearance of the sac on the mesenteric border of the intestine, in close relation to the site of perforation of the intestinal vessels.

Incidence.—Hitherto, diverticulosis of the small intestine has been regarded as a relatively uncommon condition. Radiological demonstration of diverticulosis was first performed by Case (1920), who demonstrated only 4 examples in a series of 6847 examinations (0·06 per cent). Rosedale (1935) found no case of diverticulosis in 5000 barium meal examinations. Edwards (1936) states that in a series of 2820 autopsies, only 9 cases of diverticula (0·31 per cent) were noted, and in a series of 4631 barium meals, only in 4 cases (0·086 per cent) could diverticula be diagnosed with certainty. Rankin and Martin (1934), however, who made a special study of the small intestine, demonstrated 3 examples in 956 radiological examinations (0·31 per cent).

The authors have reviewed a series of 2161 barium meal reports rendered during 1949 and 1950 and found 9 cases of proved diverticulosis of the small bowel (0·42 per cent). During the course of eighteen months 5 operations were undertaken for relief of symptoms due to this condition. In a further 4 patients it was noted at laparotomy to be present along with other pathology and it has been diagnosed in yet another 4 not operated upon. It would appear therefore that the condition is by no means a rarity, and if looked for and considered as a factor in differential diagnosis, it may come to occupy a minor but definite place among the conditions causing acute or chronic gastro-intestinal upset.

Benson, Dixon, and Waugh (1943) collected 122 cases in the Mayo clinic between 1909 and 1942 and described the condition fully. They estimated that a further 200 cases had been described in the literature. In their series, 85 were discovered at necropsy only, 21 were noted at operation, and only 16 on X-ray examination.

Site.—In Benson's Mayo clinic series, in 100 cases the jejunum only was involved, in 17 the ileum only was affected, and in 5 diverticula were found along the entire small intestine.

In the 13 patients seen by the authors, the jejunum alone was involved in 11 cases. In the remaining 2 cases, both jejunum and ileum were affected. Edwards (1936) noted 11 cases in which the jejunum alone was involved and 1 involving the ileum.

Sex and Age.—In Benson's series, 84 of the 122 cases occurred in the male sex, and Edwards, reporting 12 cases, noted the same proportion—8 males and 4 females. In both series the age incidence was comparatively high, ranging from 50 to 70 years. In the series here reported 5 were women and 8 were men and their age incidence was from 36 to 71 years. Examples in young children have been reported and will be referred to below in the discussion of pathology.

Pathology.—The morbid anatomy of small-intestine diverticulosis has been fully described by Edwards (1936). He made a detailed study of 12 specimens. He came to the conclusion that the typical example represented a herniation of the mucous membrane of the jejunum through the muscle wall of the intestine at the site of entry of the blood-vessels. In all his specimens, save one, the diverticula were on the mesenteric border of the intestine. In his one example of a diverticulum on the antimesenteric side of the intestine, the opening appeared to correspond with an unusually large blood-vessel. One of our own cases of solitary diverticulum was also on the antimesenteric side of the jejunum about 8 cm. from the duodenojejunal junction.

The blood-vessels enter the wall of the intestine on each side of and very close to the line of mesenteric attachment, and the diverticula arise at these points of penetration. They may either present as small bulges or sacs adjacent to the mesentery or as protrusions lying within the peritoneal folds of the mesentery (*Fig.* 208). This type is almost certainly acquired and the cause would appear to be the weakening of a portion of the bowel wall by the entry of blood-vessels (the ' locus minoris resistentiæ ' of Edwards) and increased intestinal pressure due to irregular and over-active peristalsis. We are in agreement with Edwards that there is no evidence that chronic intestinal obstruction plays a part in the formation of diverticula.

The smaller diverticula appear to carry on a covering of muscle-fibres, but the larger are almost completely devoid of a muscle coat and consist of

a thin covering of peritoneum lined by submucosa and mucosa (*Fig.* 209).

There are occasionally found diverticula of the small intestine which are almost certainly of congenital origin. These are often associated with congenital anomalies elsewhere, as in *Case* 13. Somerford (1930) labelled as congenital an example found in an infant in which hæmorrhage was a feature.

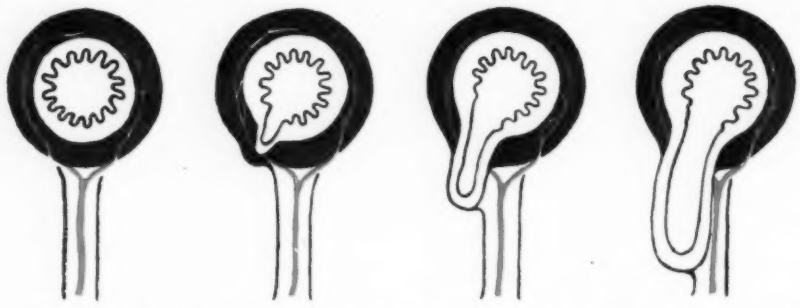

Fig. 208.—Diagram illustrating formation of a diverticulum. Note the sites of vascular penetration, the absence of mucous membrane folds in the diverticulum, and its gradual incorporation in the mesentery.

The diverticulum was solitary and arose from the mesenteric side of the jejunum. Kozinn and Jennings (1941) report the occurrence of diverticulitis in a two-year-old girl which led to peritonitis and death. This diverticulum, which was gangrenous, was the size of an adult finger and rose

FIG. 209.—*Case* 9. Low-power view of uninflamed diverticulum. (Approx. × 4·5.)

from the antimesenteric side of the jejunum. Congenital diverticula are usually solitary; they usually arise from the anti-mesenteric side of the jejunum and the muscle coat is represented in its entirety. It is possible that the solitary diverticulum reported by Edwards, and *Case* 13 in this report, may be of this nature, although from the histology Edwards considered his case to be of the acquired type. Lewis and Thyng (1908) noted the presence of diverticula and accessory epithelial nodules along the course of the œsophagus, stomach, and small intestine in pig, rabbit, and human embryos. Such nodules tend to disappear, but a few may persist and give rise to cysts and diverticula. Another explanation of the congenital diverticulum is that it is a remnant of the omphalomesenteric duct. Kozinn and Jennings think this is unlikely, owing to the variability of position of congenital diverticula.

Clinical Features.—Broadly speaking one may divide patients with diverticulosis of the small intestine into three groups: (1) Those who exhibit no symptoms at all; (2) Those who have mild chronic indigestion; (3) Those who suffer from an acute abdominal catastrophe due to a complication arising in one or a group of diverticula.

1. The frequency with which diverticula unassociated with symptoms occur is necessarily a matter of conjecture, as they may be overlooked at the time of a laparotomy where other pathology catches the eye of the operator. They are not always obvious at an autopsy as they are often deflated in death and a routine inspection of the gut may overlook them. They may well be missed during a barium meal examination, as the routine examination of the stomach and duodenum is completed before the barium has had time to fill the diverticula, and a five- or six-hour film may miss them as they have had time to empty. It is likely, therefore, that the percentage found at autopsy, operation, or on X-ray examination is far below the true figure.

2. It is true to say that a few people do suffer from vague chronic dyspepsia for which no cause can be found other than diverticulosis of the small intestine. The milder symptoms are probably due to the irregular bowel contractions which predispose towards the formation of diverticula, but the diverticula themselves may give rise to symptoms, owing to failure to empty completely, with resulting catarrhal changes within the lumen. More acute symptoms may arise if the lumen becomes blocked by catarrhal changes, or if a concretion forms and becomes impacted within a diverticulum. The symptoms may be vague and consist of flatulence, borborygmi, or upper abdominal discomfort unrelated to food or bowel action. The patient says he has "gas in the stomach" and may complain of loss of appetite. Occasionally the pain may be sufficiently severe to suggest a diagnosis of peptic ulcer or cholecystitis. It is thought that *Cases* 4, 5, 6, and 7 fall into this category. *Case* 4 continued to have occasional attacks of acute abdominal pain of unexplained origin around the umbilicus until his diverticulum was excised. *Case* 8 had occasional attacks of pain simulating peptic ulceration after the section of gut containing the bleeding diverticula had been excised. This was found on further investigation to be due possibly to more diverticula in the lower jejunum and ileum which had not been removed.

3. *Acute Complications.*—The incidence of these is low. Comprehensive tables of all the possible

complications have been set down by Milnes Walker (1945) and by Benson, Dixon, and Waugh (1943).

The major complications described are :—

a. Acute diverticulitis resulting in : (i) Perforation with generalized peritonitis or abscess formation ; (ii) Adhesions or stricture which may produce intestinal obstruction, torsion, or volvulus (d'Abreu, 1944 ; Johnson, 1945 ; Porter, 1946) ; (iii) Cyst formation where the mouth of a diverticulum is occluded by scarring following an acute inflammatory reaction (Gordinier and Sampson, 1906).

b. Hæmorrhage.

c. Concretion formation (Armitage, Fairweather, and Johnstone, 1950 ; Klidjian, 1946).

d. Traumatic rupture (Butler, 1937 ; Bagley and Bagley, 1946).

e. Foreign-body impaction.

f. Infestation with intestinal parasites (Goinard and Courrier, 1929).

Tumour formation in relation to a jejunal diverticulum was reported by Edwards, but it was by no means clear that this was a complication of the diverticulum as numerous malignant deposits were found in other sites.

Radiological Diagnosis.—Any patient suffering from vague dyspepsia, in whom the commoner forms of gastro-intestinal pathology have been excluded, should be examined with the possibility of diverticulosis being demonstrated. To see the barium filling a jejunal pouch, the meal should be followed at half-hourly intervals. If a diverticulum is demonstrated, it should be palpated under the fluorescent screen, and if tender, the evidence is strongly in favour of the diverticulum being the cause of the patient's symptoms. Attempts have been made with a Miller-Abbott tube to cause diverticula to fill by injecting barium into a segment of bowel obstructed by the inflated balloon, but this is unlikely to be of practical value.

Treatment.—

Medical Measures.—These may be adopted in cases with mild symptoms. A low-residue diet should be administered, small meals being given frequently. These should be supplemented by anti-spasmodic therapy. Some authorities have recommended a course of intestinal antiseptics such as phthalylsulphathiazole or sulphasuccidine although it is very doubtful if these can be of much value.

Surgical Measures.—These are indicated when the symptoms are more severe or in the presence of complications. Laparotomy should be performed and the diseased segment of gut located. The subsequent course to be adopted is determined by the existing pathology :—

a. Resection of gut with anastomosis is usually necessary when multiple diverticula are present in a short length of bowel ;

b. Excision of the diverticulum alone with invagination of the stump by a purse-string suture—this is satisfactory in the case of the solitary uninflamed lesion ;

c. A concretion may be broken up and expressed into the gut followed by removal of the diverticulum ;

d. A foreign body, e.g., a fish bone, may be removed through healthy gut opposite the sac and the diverticulum then dealt with.

CASE REPORTS

These may be divided into : (*a*) Those exhibiting no symptoms (*Cases* 1, 2, 3) ; (*b*) Those who have mild chronic indigestion (*Cases* 4, 5, 6, 7) ; (*c*) Those with acute complications (*Cases* 8, 9, 10, 11, 12, 13).

Case 1.—Mr. F. C., aged 66.

HISTORY.—The patient had suffered from severe indigestion for some ten years. He complained of severe pain coming on one hour after meals. It was situated mainly in the epigastrium, but tended to radiate to the left side of the abdomen. It was relieved by ingestion of milky foods and by alkalis. In addition he complained of nausea with occasional bilious vomiting, which also relieved his pain. He was troubled greatly with flatulence. His appetite was poor, but he stated that he had lost no weight.

His appendix had been removed some years previously.

ON EXAMINATION (Nov. 8, 1950).—The patient's general condition was satisfactory, but abdominal examination revealed definite tenderness in the region of the duodenum.

A barium meal was given and the radiologist reported as follows : "The stomach is normal and empties normally. There is deformity of the duodenal cap and a small crater is demonstrated. He is tender over the cap. He has a congenital diverticulum of the second part of the duodenum and a few in the upper part of the small bowel. There is normal passage through the small bowel and colon."

Surgical treatment, primarily for the duodenal ulcer, has been recommended. It is unlikely that the diverticula are responsible for his symptoms.

Case 2.—Mrs. M. R., aged 35.

HISTORY.—The patient had complained of periodic attacks of epigastric pain with associated heartburn and waterbrash for some eighteen months. There was no apparent relationship to ingestion of food, but alkalis gave some relief. She occassionally vomited small amounts of bile. She thought she had lost a little weight.

ON EXAMINATION.—The patient was noted to be very thin. The abdomen was scaphoid, but exhibited no tenderness, guarding, or other abnormality. Rectal examination was negative.

Barium meal : the duodenal cap was shrunk, suggesting distortion due to an old ulcer. The gastric emptying rate was slow, but there was no residue of barium at twenty-four hours. A diverticulum was noted in the upper jejunum ; this too was empty at twenty-four hours.

AT OPERATION (July 20, 1950).—The duodenum was found to be densely scarred, and partial gastrectomy (Polya) was carried out. The appendix was coiled and adherent and was removed. Two large diverticula, one approximately 3 × 3 cm. and the other 2 × 2 cm., were found on the mesenteric border of the first coil of jejunum and were removed by excising 15 cm. of gut, end-to-end anastomosis being performed.

The post-operative convalescence was uneventful.

Histological examination revealed the features of the pulsion type of jejunal diverticulum, the wall consisting only of mucosa, submucosa, with muscularis mucosa and serosa.

Case 3.—Mrs. E. M., aged 59.

HISTORY.—The patient complained of vague upper abdominal pain with a smaller amount in the right iliac fossa. She attributed the pain to an accident she had had five years previously. More recently she had begun to vomit small amounts of clear fluid like water, the vomiting relieving the pain. She had lost some weight, but had always been rather thin.

ON EXAMINATION.—The patient was a rather small, thin, pale woman exhibiting definite tenderness in the epigastrium and deep to the lower right rectus. The

liver was thought to be slightly enlarged. A barium meal was given. The patient would only take 6 oz. of barium as she felt she would be sick. There was noted to be some irregularity of the lesser curve and posterior wall of the stomach. The appendix could not be visualized and there was marked tenderness over it. The examination was considered to be inconclusive owing to the small amount of barium taken, but the radiologist suggested gastroscopy to exclude carcinoma of the stomach.

AT OPERATION (Nov. 13, 1950).—There was found to be an old scarred duodenal ulcer with pyloric stenosis and an active ulcer on the lesser curve of the stomach. A solitary diverticulum was present on the mesenteric border of the highest coil of the jejunum.

Partial gastrectomy (anterior Polya type) with local excision of the diverticulum was carried out.

Convalescence was uneventful and the patient was discharged in a greatly improved condition.

Case 4.—Mr. G. P., aged 57.

HISTORY.—The patient was operated upon in May, 1948, when partial gastrectomy for an ulcer on the posterior wall of the first part of the duodenum was carried out. At this intervention it was noted that there were some diverticula in the duodenum but no description was made of their size or exact position.

He recovered satisfactorily following this intervention, but continued to have mild symptoms—a sense of fullness, flatulence, and nausea with occasional slight bilious regurgitation.

A barium meal was carried out in February, 1949. This revealed a normally functioning stoma with a portion of gut distended with gas and containing a fluid level lying just medial to the stoma. It was suggested by the radiologist that it might be an acquired diverticulum.

As the patient's symptoms persisted, it was decided to re-explore the abdomen and perform diverticulectomy.

AT OPERATION (June 28).—The gastric remnant, the stoma, and the duodenal stump appeared satisfactory. On the afferent loop, just proximal to the anastomosis, a diverticulum extended from the mesenteric border of the gut between the layers of the mesentery. It was about 4 cm. in length. There was no evidence of complication. Treatment was limited to simple resection of the diverticulum.

HISTOLOGY.—This revealed a simple diverticulum without unusual features.

The patient subsequently stated that he felt very much improved. When last seen (Oct. 23, 1950) he stated that he still had occasional nausea if he took other than his ordinary diet, but that generally his condition was satisfactory.

Case 5.—Mrs. W. O'C., aged 58.

HISTORY.—The patient gave a very vague history of abdominal discomfort and nausea, with some associated low backache. She thought that she might have lost some weight. Her bowels usually tended to be constipated, but she had had an attack of diarrhœa a few weeks previously, although this could have been related to some epidemic. She thought her symptoms might be attributable to 'nerves'.

ON EXAMINATION (June 7, 1950).—The tongue was perfectly clean. Abdominal examination revealed no tenderness, swelling, or other abnormality. Rectal examination was negative.

A barium meal report was as follows: "The stomach shows normal appearances and active peristalsis and empties normally. The cap shows slight deformity, but no ulcer crater can be demonstrated. It is not tender. There is normal passage of the barium through small bowel and colon. The appendix is seen filled. Several diverticula are shown in the jejunum. These do not empty with the passage of the meal and are seen to persist some hours after the meal has passed into the colon."

As the patient's symptoms were mild it was decided to keep her under observation, appropriate dietary measures being prescribed.

Case 6.—Mrs. G. R., aged 42.

HISTORY.—The patient gave a history of having had a vague feeling of discomfort in the upper abdomen for some months. She described it as being a hungry feeling which was not relieved but rather aggravated by food. Alkalis produced some improvement. Apart from this she was symptom-free.

ON EXAMINATION (June 7, 1950).—The patient was a healthy-looking woman. Her tongue was clean and the breath inoffensive. Abdominal examination was negative apart from the presence of slight tenderness in the left iliac fossa.

A barium meal was given. The stomach and duodenum were found to be normal, but a large diverticulum was seen near the duodenojejunal flexure.

On re-examination (July 12) there was found to be definite abdominal tenderness situated just to the left of the umbilicus. It was considered that this solitary clinical finding could be explained by the radiological finding of the large diverticulum in this region.

The patient did not feel that her symptoms were sufficiently severe to warrant surgical intervention. She is being kept under observation.

Case 7.—Mr. H. J., aged 40.

HISTORY.—The patient presented himself in November, 1949, with a complaint of over five years' stomach trouble, for which he had previously had medical treatment in hospital. He suffered from pain after food, hunger pain, and night pain, relief being afforded by food, alkalis, and vomiting. He had lost some weight. There were periodic intermissions in the symptoms.

Radiological examination in 1947 when in the Forces had suggested the presence of a juxta-pyloric ulcer. In December, 1949, the radiologist reported the presence of diverticula in the stomach, the second part of the duodenum, and in the jejunum.

ON EXAMINATION.—The patient was noted to be nervous. The abdomen revealed only slight epigastric tenderness.

AT OPERATION (Jan. 10, 1950).—There was scarring of the duodenum, indicating ulceration, and partial gastrectomy was carried out. Diverticula were noted in the upper jejunum, with multiple adhesions especially around the gall-bladder, which, however, appeared to be normal.

Following this intervention the patient was well for a time, but subsequently his old symptoms returned. A repeat barium meal revealed a normally functioning stoma.

It was considered that a large psychological element was present, but it was decided to carry out diverticulectomy.

AT OPERATION (Aug. 8).—A large pulsion diverticulum showing no evidence of complication was excised from the afferent loop of the jejunum. A similar diverticulum, but surrounded by adhesions and showing evidence of chronic inflammation, was found in the terminal ileum and was also excised.

Histological examination confirmed the presence of chronic diverticulosis in the ileal diverticulum, there being very marked round-cell infiltration.

FOLLOW-UP.—After a further period of relief the patient's symptoms have returned, although they are now much less severe. Treatment is being continued with medical measures.

Case 8.—Mrs. S. W., aged 71.

HISTORY.—The patient was admitted to hospital after a hæmatemesis of moderate severity. This had been followed by melæna. She stated that she had been well until one year previously, when she began to lose her appetite. She had had no pain.

On Examination.—The patient was a frail-looking woman who had obviously lost a lot of weight. Abdominal examination was negative, although the patient's doctor reported that at the time of the bleeding there was some tenderness in the right flank. Rectal examination revealed nothing abnormal and there was no blood on the examining finger. A provisional diagnosis of carcinoma of the stomach was made and a barium meal was carried out. This showed no evidence of gastric carcinoma but several round dense masses of barium were noted in the follow-through films which suggested the diagnosis of jejunal diverticula.

At Operation (April 5, 1949).—The stomach and duodenum were found to be normal. Multiple diverticula were found on the first 2 ft. of the jejunum, lying between the layers of the mesentery. They did not present any unusual features. The affected segment of bowel was resected and bowel continuity restored by end-to-end anastomosis.

Naked-eye examination of the resected gut revealed six diverticula from 1½ to 4 cm. in diameter. They were all on the mesenteric border at intervals of 5 to 6 cm. (*Fig.* 210). Histological examination of one diverticulum showed the wall to consist of mucosa, submucosa, muscularis mucosa, and serosa with no muscularis. There was marked round-cell infiltration, including eosinophils, into the mucosa. The herniation was through the muscle coat of the bowel.

Convalescence after operation was smooth and the patient remained well until some nine months after operation, when she had a recurrence of epigastric pain with vomiting. A suggestion of a small gastric ulcer was revealed by barium meal, but a repeat examination revealed a perfectly normal stomach. It was noted, however, that small residues of barium were present in the region of the jejunum and it was suggested that there might be small residual diverticula.

The latest report from the patient is that she is now symptom-free.

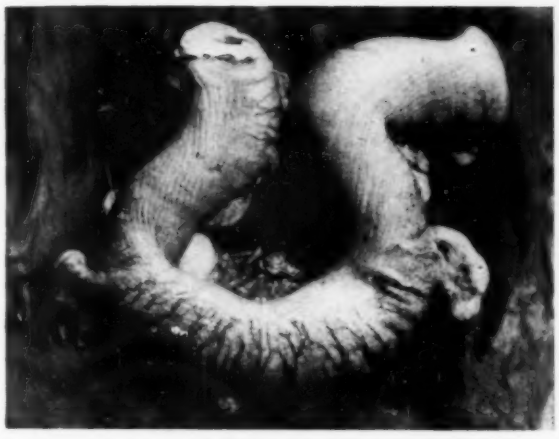

Fig. 210.—Hæmorrhage from jejunal diverticula. Resected specimen in *Case* 8.

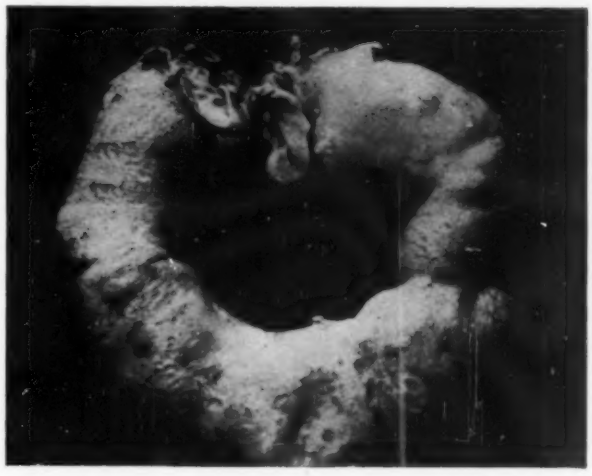

Fig. 211.—*Case* 9. Acute jejunal diverticulitis showing subperitoneal exudate.

Case 9.—Mr. W. L., aged 46.

History.—The patient was suddenly seized with acute abdominal pain while at work on the morning of the day of admission. On arrival at hospital the pain was still present as a constant ache across the abdomen, but it was now maximal in the left iliac fossa. He had felt nauseated but had not vomited. He had, however, been sick on two occasions on the previous day, but apart from this incident there was no history of indigestion.

On Examination.—The patient was a well-nourished heavily-built man. He was obviously in severe pain. The pulse-rate was elevated to 120 and the temperature to 98·6° F. His tongue was lightly coated. The abdomen exhibited very definite tenderness and guarding down the whole of the left side and extending well round into the left flank. There was very marked release tenderness. Bowel sounds were present. There was no loss of liver dullness. Urinary examination was negative. Straight radiographs revealed no subphrenic gas and no evidence of renal calculus or other abnormality.

Fig. 212.—*Case* 9. Acute jejunal diverticulitis. Orifice of diverticulum.

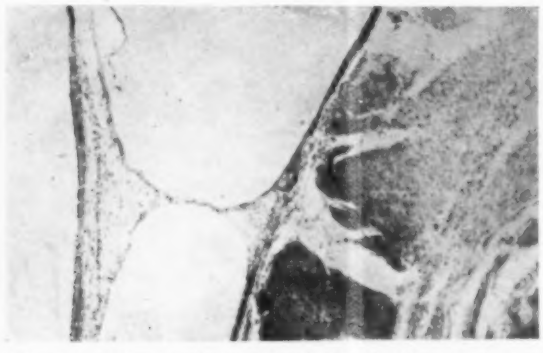

Fig. 213.—*Case* 9. Acute jejunal diverticulitis—exudate. (× 37·5.)

AT OPERATION (April 6, 1949).—There was a little free seropurulent fluid in the peritoneum. Marked thickening was present over a length of about 25 cm. of the upper jejunum, some 2 ft. below its commencement. There

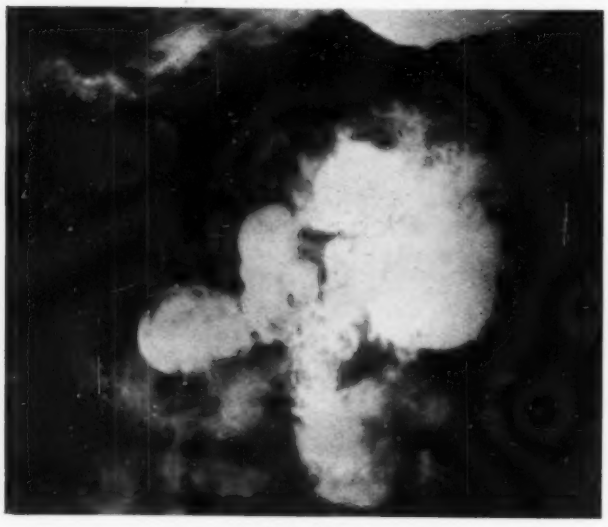

FIG. 214.—*Case* 10. Radiological demonstration of jejunal diverticulosis.

was a considerable amount of pus in the subserosal layer and the surface was elevated in parts by numerous, small pearly bubbles of imprisoned gas. Larger bubbles were present between the layers of the mesentery, which was very markedly thickened and œdematous. Towards

FIG. 216.—Hæmorrhage from jejunal diverticula. Resected specimen in *Case* 10.

the centre of the mass a diverticulum was found to extend for a distance of about 3 cm. into the mesentery. A small uninflamed diverticulum was present in a similar position, about 15 cm. lower in the jejunum. About 30 cm. of jejunum was resected and an end-to-end anastomosis performed (*Fig.* 211).

Histological examination confirmed the presence of an acute inflammatory reaction in the larger diverticulum (*Figs.* 212, 213).

CONVALESCENCE.—This was uneventful. Three months after operation the patient was very fit.

Case 10.—Mr. W. H., aged 54.

HISTORY.—The patient had complained of moderately severe dyspepsia for some 22 years. He stated that he had vomited 'coffee-grounds' as far back as 1928. X-ray examination at that time had revealed an irritable, rapidly emptying stomach and duodenum, but did not demonstrate an ulcer crater. In 1937, when he was X-rayed again, a very similar report was forthcoming, but in addition a very extensive diverticulosis of the large gut was demonstrated with what appeared to be a diverticulum high up in the jejunum.

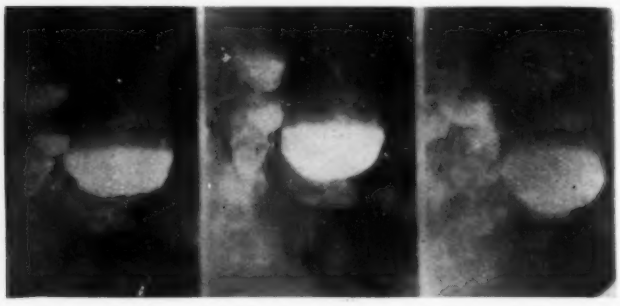

FIG. 215.—*Case* 10. Serial radiographs using compression.

A repeat radiological examination in March, 1950, following a fresh hæmatemesis, confirmed the presence of jejunal diverticulosis (*Figs.* 214, 215). No pathology in the œsophagus, stomach, or duodenum was found which could have accounted for the bleeding.

FIG. 217.—*Case* 10. Section through diverticulum.

A pre-operative insulin test-meal revealed almost complete achlorhydria. The night secretion test showed complete absence of free acid with a total acid value of 15 units.

AT OPERATION (March 28, 1950).—There was no evidence of pathology in the stomach or duodenum, but four large diverticula were found in the highest loop of the jejunum. The largest was about 4½ cm. in diameter. They all lay along the mesenteric border of the gut,

projecting between the layers of the mesentery (*Figs.* 216, 217). A segment of jejunum about 20 cm. in length was excised, the upper line of section passing through the duodenojejunal flexure. The gut was reconstituted by end-to-end anastomosis.

Histological examination of the largest diverticulum revealed the typical structure of a pulsion diverticulum with absent muscularis. The mucosa was noted to be densely infiltrated with round cells.

The patient's convalescence was uneventful and he was discharged home twelve days after operation.

His digestion has since been greatly improved and there has been no recurrence of hæmatemesis.

Case 11.—Mr. R. I., aged 64.

HISTORY.—The patient had suffered from indigestion for about twenty years, the main features being pain after meals, hunger and night pain, and vomiting. The pain was relieved by food, by alkalis, and by vomiting. There was marked periodicity of the symptoms. He had had three hæmatemeses, but no melæna had been noticed.

ON EXAMINATION.—There was very little to be made out clinically in the abdomen, but the radiographs revealed numerous diverticula in the second part of the duodenum and the small and large bowel (*Fig.* 218). The stomach and first part of the duodenum showed normal appearances and emptied normally.

AT OPERATION (July 1, 1950).—The stomach, duodenum, and gall-bladder were normal. There were multiple diverticula in the upper 3 ft. of the jejunum, varying in size up to that of a golf ball. They were all lying on one or other side at the mesenteric attachment of the gut. There was no thickening or evidence of complication. Further inspection of the small bowel revealed the presence of a thickening in the terminal ileum about 40 cm. from the ileocæcal valve. There was evidence of some narrowing of the lumen, with partial obstruction, and the gut above showed moderate distension. The large gut was normal apart from slight diverticulosis in the sigmoid. It was considered that some at least of the patient's symptoms could be attributed to the definite pathology in the ileum, although this could not have accounted for the hæmorrhage, and the operation was limited to resection of about 20 cm. of ileum with end-to-end anastomosis.

PROGRESS.—Convalescence was uneventful.

HISTOLOGY.—This revealed a chronic ileitis. There was no endothelial or giant-cell reaction. It was not possible to say whether or not it was a late stage of Crohn's disease.

Case 12.—Mr. S. W., aged 56.

HISTORY.—The patient was admitted to the medical wards after vomiting a small amount of altered blood. He gave an old history of a duodenal ulcer and had been operated on some eight years previously when a gastroenterostomy was performed. He had had one hæmatemesis after this, but had been symptom-free for the last six years.

PROGRESS.—There was no recurrence of hæmatemesis after admission, and two weeks later a barium meal was given. The radiologist reported: "The stomach was about half full before the stoma commenced to function. Shortly afterwards some barium passed through the pylorus. There was no tenderness over the stoma and no ulcer crater was demonstrated. The stomach showed a normal emptying rate. Two diverticula of the small intestine were shown."

AT GASTROSCOPY (Nov. 9, 1950).—A good view was obtained. The gastric mucosa and stoma appeared quite satisfactory. There was no evidence of ulceration.

It was recommended that laparotomy should be performed in the event of a recurrence of hæmorrhage. While it was considered probable that the bleeding was the result of erosion, either in the duodenum or at the stoma, it was felt that the jejunal diverticula could not be completely ruled out as the source, particularly as his recent hæmatemesis had not been associated with pain or abdominal tenderness.

Case 13.—Mr. E. R., aged 20.

HISTORY.—The patient was admitted to hospital as an acute emergency. He complained of severe but

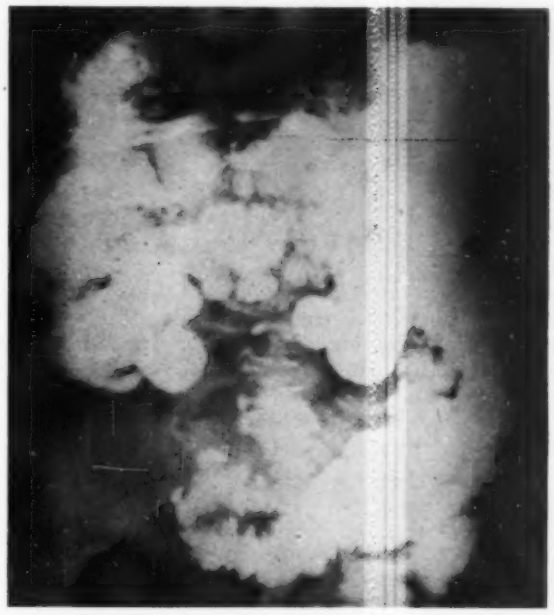

FIG. 218.—*Case* 11. Radiological demonstration of jejunal diverticula.

intermittent epigastric pain of some five hours' duration, with associated vomiting. The pain had become much more severe a short time before admission. He gave a previous history of epigastric pain after meals, but this had never been severe.

ON EXAMINATION.—The pulse was 100 and the temperature normal. The abdomen was markedly guarded in all areas and was silent. There was no loss of liver dullness. Rectal examination revealed marked tenderness in the pelvis.

With a provisional diagnosis of appendicitis it was decided to explore the abdomen.

AT OPERATION (Sept. 12, 1950).—A right gridiron incision was made. The cæcum could not be found. Coils of jejunum presented in the wound and on one of these a diverticulum was noticed. The gridiron incision was closed and the abdomen re-opened by a right paramedian incision.

There was very evident failure of rotation of the gut, the whole colon lying in the left iliac fossa and pelvis, suspended by a long mesentery; with the second part of the duodenum leading straight down into the jejunum, which lay on the right side of the abdomen. The diverticulum was found to be situated about 8 cm. below the duodenojejunal junction. It lay on the antimesenteric border, projecting for a distance about equal to the width of the gut. A narrow fold of peritoneum with a blood-vessel along its free margin passed from the apex of the diverticulum for a short distance up the gut and a longer distance (about 20 cm.) downwards along the antimesenteric border. An enlarged lymph-node lay between the layers of this fold.

Treatment was limited to local excision of the diverticulum, appendicectomy, and removal of the solitary lymph-node for biopsy.

The patient's convalescence was smooth, apart from a complaint of slight pain, similar to his previous pain, on the eighth post-operative day and again one month later.

HISTOLOGICAL EXAMINATION revealed the diverticulum to consist of normal intestinal mucosa, submucosa, muscularis, and serosa, the mucosa and submucosa being heavily infiltrated with chronic inflammatory exudate. The lymph-node showed reactionary inflammatory hyperplasia.

DISCUSSION

Among the cases reported there are several in which the diverticula have been associated with severe symptoms. In some instances urgent operation has been necessary.

Four patients sustained repeated attacks of hæmatemesis for which no cause other than diverticula was discovered. In two of these patients resection was carried out for the relief of hæmatemesis, with satisfactory results. The remainder are being watched carefully for evidence of further bleeding, with the possibility that resection of a segment of affected gut or a diverticulum may, in the absence of other pathology, be indicated. Hitherto only 9 cases of hæmatemesis or melæna from diverticula of the jejunum have been reported in the literature—namely, by Braithwaite (1923), Somerford (1930), Tengwall (1931), Haltung, reported by Rankin and Martin (1934), Jones and Crile (1936), Guthrie and Hughes (1937), Normark (1938), Klidjian (1946), and by Kozoll, McMahon, and Kiely (1950). In Braithwaite's case there was severe hæmatemesis. Somerford's case has already been mentioned. Normark described a case in a child aged 2 years. In this there was severe hæmorrhage, which at laparotomy was found to originate in a large non-Meckelian diverticulum of the small intestine. This was almost in the form of a duplication of a short length of the gut, and ran lengthwise between the layers of the mesentery, one end opening into the bowel and the other ending in a narrowed portion resembling the vermiform appendix. Uneventful recovery took place after resection. Klidjian's patient, a man of 73, exhibited the complications of concretion formation and severe hæmorrhage into the gut. Eight diverticula were present. The largest contained a stony-hard concretion and was discoloured, while the surrounding mesentery was congested. It appeared likely that the hæmorrhage took place from this diverticulum. The last-mentioned authors reported a most convincing case in a man of 60. He presented with severe and repeated melæna and it was estimated that some 16,000 ml. of blood were lost into the gut. Exploration after massive transfusion revealed extensive diverticulosis of the jejunum. Practically the entire jejunum was resected and the patient recovered. Naked-eye examination of the diverticula revealed numerous erosions.

It is considered that the authors' two proven cases of hæmorrhage may be added to the 9 already reported. It should be noted that, as in our cases, the blood may be vomited owing to reverse peristalsis, although melæna has been the more common manifestation in cases previously reported.

Two patients presenting symptoms of acute abdominal inflammation have been found to suffer from diverticulitis. In one case a solitary congenital diverticulum of the jejunum was inflamed, and in the other, which was associated with severe toxæmia and peritonitis, a pulsion diverticulum was affected. In a third case a chronically inflamed diverticulum was excised from the terminal ileum. Although a more frequent complication than hæmorrhage, the occurrence of acute diverticulitis is also rare. Milnes Walker (1945) stated that 19 cases had been recorded, and added a further 3 cases. Benson, Dixon, and Waugh (1943), in their review of 122 cases of jejunal diverticulosis, cited only 1 case in which there was diverticulitis with perforation, a further 2 cases examined post mortem showing only mild catarrhal inflammation.

In 5 cases the presence of diverticula appears to have been coincident with duodenal ulcer, and the diverticula are probably not responsible for the patient's symptoms. Irregular peristalsis is recognized as one of the factors leading to the formation of the acquired or pulsion type of diverticulum, and it is accordingly interesting to speculate whether the vagotonia, so commonly associated with duodenal ulcer, may also play a part in the production of pulsion diverticula.

All but one of the cases reported present diverticula of the acquired type, *Case* 13 being the only example of a diverticulum of congenital origin. A survey of the literature reveals that such diverticula are excessively rare.

The authors would suggest in conclusion that diverticulosis of the jejunum is by no means a rarity, that it may be the cause of hæmatemesis or melæna, that it may give rise to acute abdominal symptoms, or may be a cause of chronic vague dyspepsia. When other pathology can be excluded, diverticulectomy or resection of the affected segment of jejunum may be expected to afford relief.

SUMMARY

1. A review is made of diverticulosis of the jejunum emphasizing its relative frequency.

2. Thirteen patients with jejunal diverticulosis are reported.

3. The pathology, complications, with special reference to hæmorrhage and inflammation, and treatment are discussed.

We wish to express our thanks to Mr. R. Cunliffe Shaw for permission to publish records of three patients who were under his charge; to Dr. A. A. Miller for the pathological studies; to the staff of the X-ray Department of the Preston Royal Infirmary for their co-operation; and to Mr. I. B. Smith for many helpful suggestions.

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THE DIFFERENTIAL DIAGNOSIS OF MALIGNANT DISEASE OF THE PERITONEUM*

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MALIGNANT disease of the peritoneum may be found at laparotomy or during the course of operations for hernia. It can usually be correctly diagnosed from its naked-eye appearance, but there are occasions in which a confident diagnosis cannot be made on the basis of operative findings alone.

This paper contains an account of the pathology of the condition, discusses the differential diagnosis, and presents 7 cases illustrating some of the diagnostic difficulties that may be encountered.

FIG. 219.—*Case* 1. Nodule from peritoneal surface of small intestine consisting of cellular fibro-fatty tissue with foci of lymphocytes, but no apparent malignant cells. On naked-eye examination it appeared as a round white nodule 3 mm. in diameter. (× 52·5.)

CASE REPORTS

Case 1.—**Stricture of the ileum and peritoneal nodules secondary to carcinoma of the stomach.** A man, aged 47, was admitted complaining of symptoms suggestive of acute small-intestine obstruction. During the previous month he had complained of epigastric pain half an hour after meals, anorexia, and loss of weight. Emergency laparotomy disclosed a cons[illegible]rable amount of ascites and a fibrous 'ring' stricture [illegible]of the ileum, 5 cm. proximal to the ileocæcal valve, c[illegible]ing obstruction. The peritoneum covering the sma[illegible] intestine and its mesentery was studded with a lar[illegible] number of round white nodules 2–3 mm. in diam[illegible]er and there were many firm lymph-nodes 1–2 cm. [illegible] diameter in the base of the mesentery. No abnorm[illegible]ity was seen or felt in the stomach or any other ab[illegible]ominal organ.

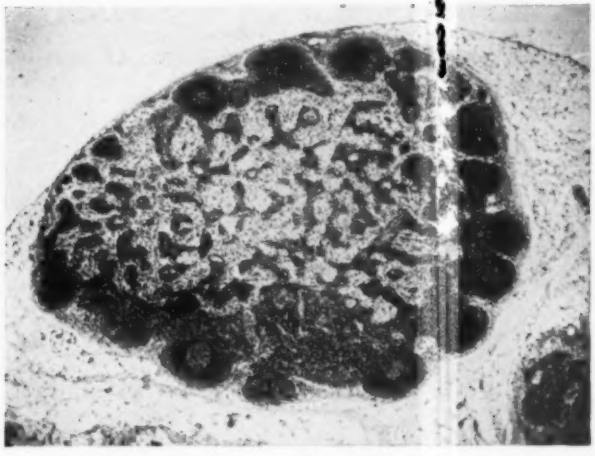

FIG. 220.—*Case* 1. Lymph-node from mesentery of small intestine, showing sinus dilatation and reticulum-cell hyperplasia but no specific changes. (× 22·5.)

The condition was considered to be either tuberculous or cancerous.

Ileotransverse colostomy was performed and a peritoneal nodule and two enlarged lymph-nodes were removed for histological study. The peritoneal nodule was later reported to be a mass of cellular fibro-fatty tissue containing perivascular foci of lymphocytes (*Fig.* 219) and the two lymph-nodes as showing marked sinus dilatation and reticulum-cell hyperplasia (*Fig.* 220). There was no evidence of cancer or tuberculosis in any of the specimens.

At autopsy, a fortnight after the operation, the immediate cause of death was found to be bronchopneumonia. The stomach appeared normal until it was opened; only then was a small, soft polypoid tumour

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found in the fundus, while the whole of the submucosal layer was seen to be infiltrated with white, gelatinous tumour tissue. Histological examination showed the

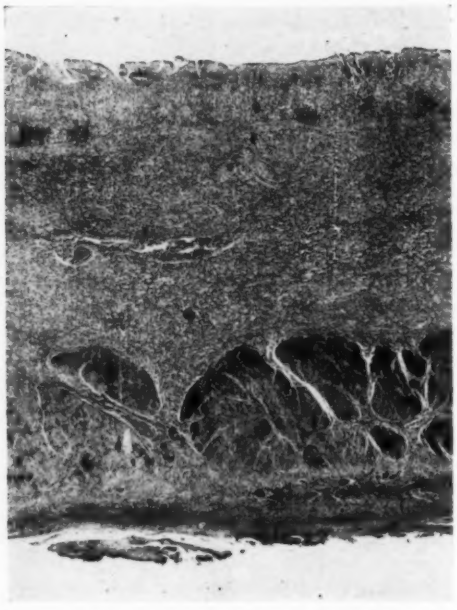

FIG. 221.—*Case* 1. Section of stomach obtained at autopsy. The mucosa has been destroyed. All the other layers of the stomach are thickened by an infiltration with cells, which under high power were found to be mucus-containing carcinoma cells. Groups of carcinoma cells are loosely attached to the outer aspect of the serosa and are presumably ready to fall into the peritoneal cavity. (× 9·5.)

polypoid mass to consist of a spheroidal-celled carcinoma with many signet-ring cells, and the whole stomach wall to be infiltrated with similar cells (*Fig.* 221). The

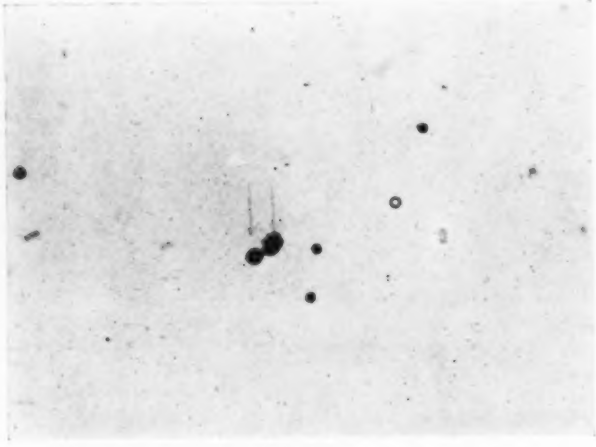

FIG. 222.—*Case* 2. Cellular deposit from ascitic fluid stained by Southgate's mucicarmine technique. The two cells marked by arrows stained bright red. In this photograph the red colour appears black. (× 215.)

stricture in the ileum showed infiltration with mucin-secreting cancer cells and extensive fibrosis.

Comment.—Gastric primary not found by laparotomy or biopsy.

Case 2.—**Tuberculosis simulating carcinomatosis of the peritoneum.**—A woman of 58 complained of progressive distension of the abdomen, loss of energy, and loss of appetite for two months. The abdominal distension was due mainly to ascites and partly to a large ill-defined tumour in the epigastrium. Multiple sinuses opened on both sides of her neck. These were surrounded by unhealthy, bluish-red skin, appeared to arise from enlarged deep cervical lymph-nodes, and had been present for ten years.

The ascitic fluid was found to contain some cells which were associated either with the production or phagocytosis of mucus. None of the cells could be reported as malignant, although the presence of mucus was strongly suggestive of a mucus-secreting carcinoma (*Fig.* 222).

At laparotomy masses of clear jelly-like material were found floating in a considerable amount of straw-coloured ascitic fluid. The epigastric tumour was enveloped in great omentum and the stomach and liver could not be defined. Numerous white nodules, 3–4 mm. in diameter, studded the peritoneum covering the large and small intestines.

Microscopical examination of a peritoneal nodule and an enlarged cervical lymph-node showed typical tuberculosis (*Fig.* 223).

Comment.—Diagnosis of the abdominal disease depended upon laparotomy and biopsy, although the presence of tuberculous cervical adenitis was clinically suggestive.

Case 3.—**Encapsulated food particles simulating peritoneal carcinomatosis.**—A man of 45 was admitted for investigation of dyspepsia. He had previously suffered several attacks of severe upper abdominal pain but had had no operation. At laparotomy a large fixed ulcer was found in the middle of the posterior wall of the stomach. The supra-pancreatic lymph-nodes were enlarged and hard and there were many firm, round, white nodules, 3 to 5 mm. in diameter, studding the pelvic floor, the mesentery of the small bowel, and the surface of the great omentum. The nodules were considered to be malignant seedlings from a carcinoma of the stomach, and the abdomen was closed after removing an omental nodule for microscopical examination. This

FIG. 223.—*Case* 2. Nodule removed from peritoneal surface of large bowel, showing typical tuberculous structure. (× 40.)

nodule was later reported to consist of bile-stained vegetable matter surrounded by chronic inflammatory tissue rich in foreign-body giant cells (*Fig.* 224). The patient returned to hospital two months later for partial gastrectomy and has since made an uninterrupted recovery. Histological examination of the excised stomach ulcer showed it to be benign.

Comment.—It is presumed that at some time this man had a perforated peptic ulcer from which he

recovered without operation. Diagnosis could be made only on histology.

Case 4.—**Fibrous nodules in a long-standing hernia simulating carcinoma.**—A man of 74 was admitted suffering from a strangulated inguinal hernia.

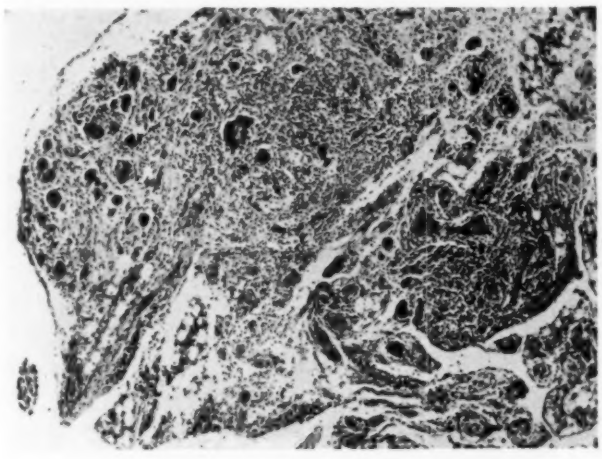

FIG. 224.—*Case* 3. Nodule from great omentum consisting of chronic inflammatory tissue rich in foreign-body giant cells. (× 40.)

There was no history of previous illness of note, or of operation. At emergency operation the hernial sac was found to contain blood-stained fluid and a partially strangulated loop of small intestine, on the surface of which were several hard nodules of slightly translucent greyish material 5 × 2 mm. in size. The nodules were thought to be metastases, probably from a colloid carcinoma. The patient made an uninterrupted recovery and now, over a year later, shows no sign of cancer (*Fig.* 225).

Comment.—Even histological examination is not always conclusive. A bad prognosis should not be given unless the evidence for it is definite.

FIG. 225.—*Case* 4. Nodule from the peritoneal surface of a loop of small intestine which was incarcerated in a direct inguinal hernia. It consists of cellular connective tissue. (× 40.)

Case 5.—**Peritoneal carcinomatosis presenting as a strangulated hernia.**—A woman of 62 was admitted complaining of intermittent attacks of abdominal pain for three months. For twenty-four hours the pain had been very severe and accompanied by repeated vomiting, constipation, and inability to pass flatus. She was obese and had a small, tense, dusky, umbilical hernia which

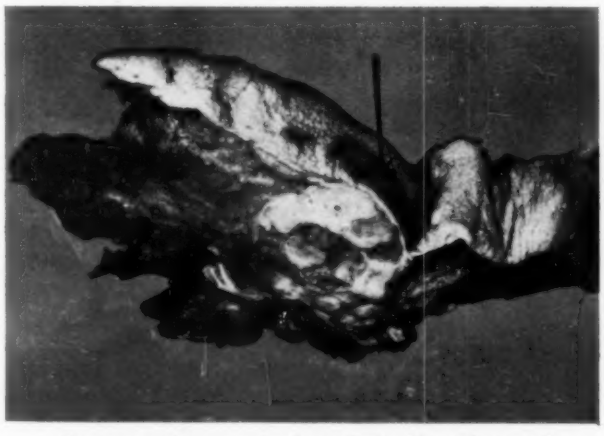

FIG. 226.—*Case* 5. A view of the bisected operation specimen showing the dusky umbilical swelling (arrow) formed by a hernial sac full of omentum in which there are nodules of white tumour. (× 0·75.)

carcinoma of the alimentary tract. A nodule was excised and later reported to consist of rather hyaline, cellular connective tissue such as is often seen in the reaction to early carcinomatous deposits. There were also present in the nodule a number of large irregular deeply staining cells which could be carcinoma cells, although it was difficult to be certain of this. The mucin which so often identifies carcinoma cells, especially those from the alimentary tract, could not be demonstrated and examination with the polarizing microscope revealed nothing of note. The final opinion of the histopathologist was that this was probably, but by no means certainly, a

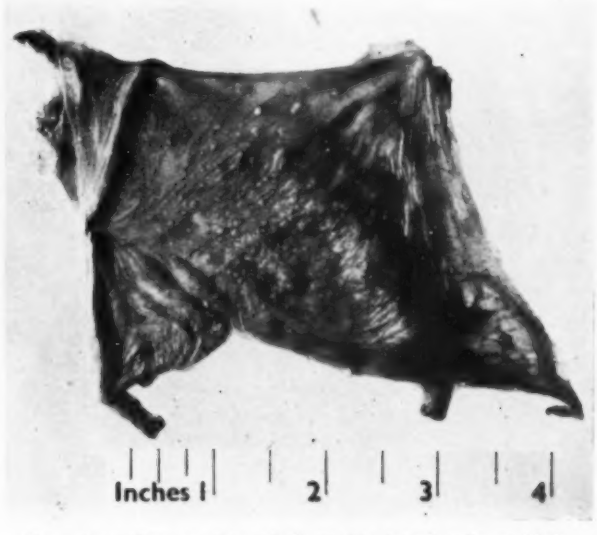

FIG. 227.—*Case* 6. Sac of direct inguinal hernia studded with white nodules.

was considered to be strangulated. At operation the hernial sac contained omentum in which were nodules of white tumour, while the abdominal cavity revealed widespread carcinomatosis (*Fig.* 226). Microscopy of the omental tumour showed it to be a papillary adenocarcinoma probably of ovarian origin.

Comment.—Peritoneal metastases may first manifest themselves through changes in a hernial sac.

Case 6.—**Encapsulated food material simulating metastases in a hernial sac.**—A man of 59 was admitted for repair of a large direct inguinal hernia. A year before he had been operated on for a perforated peptic ulcer. The excised hernial sac was found to be studded with many white nodules which were believed

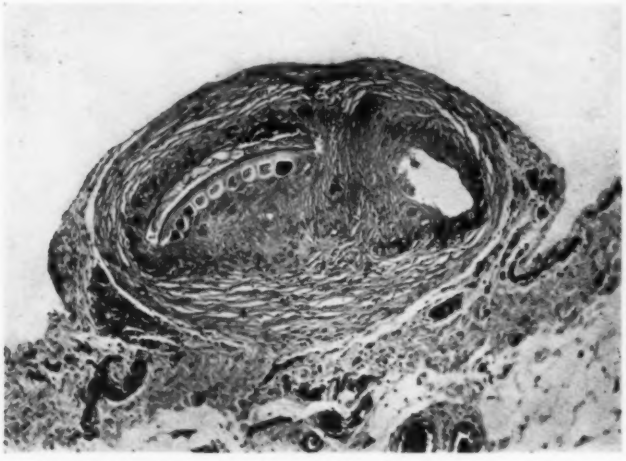

FIG. 228.—*Case* 6. Section of nodule from hernial sac showing encapsulated vegetable matter. (× 42·5.)

at operation to be either encapsulated foreign bodies or carcinomatous deposits (*Fig.* 227). Histological examination showed the nodules to consist of encapsulated pieces of vegetable matter surrounded by a mononuclear and giant-cell reaction (*Fig.* 228). The polarizing microscope demonstrated very clearly a central sliver of doubly refractile material (*Fig.* 229).

Comment.—Histological examination was required for the detection of encapsulated food material.

Case 7.—**Polyarteritis nodosa as a cause of subperitoneal nodules.**—A man of 42 was admitted complaining of nocturnal dyspnœa and œdema of the

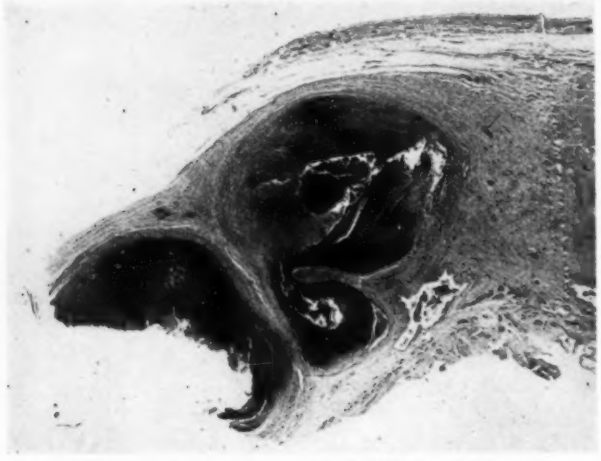

FIG. 230.—*Case* 7. Section of gall-bladder. The nodules are formed of greatly dilated tortuous blood-vessels which are full of thrombus. (× 7.)

ankles for six months. His blood-pressure was 220/150, there was evidence of cardiac hypertrophy, and his fundi showed silver wiring and slight nipping. He was afebrile and there was no leucocytosis. His urine contained a trace of albumin, but no pus cells. Intravenous urography and urea-clearance tests showed normal renal function.

Lumbodorsal sympathectomy was performed on the left side through a transpleural approach. Seventeen days later he complained of severe pain in the left hypochondrium and showed signs of collapse. Emergency laparotomy revealed a considerable quantity of blood-clot in the left upper abdomen and a bleeding point

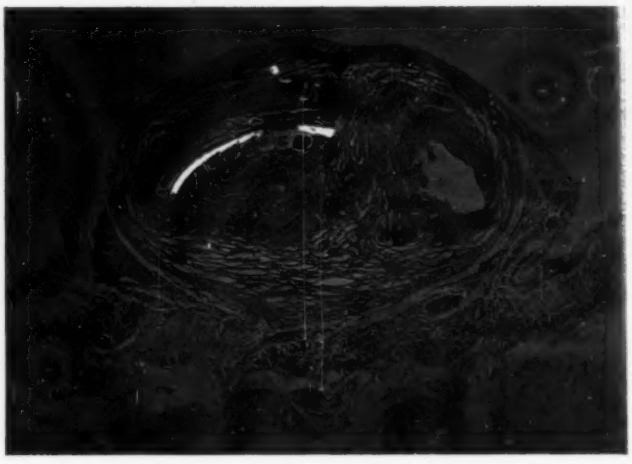

FIG. 229.—*Case* 6. Section of nodule from hernial sac photographed in polarized light. The central stalk of the encapsulated vegetable matter appears as a bright white streak. (× 42·5.)

in the hilum of the spleen. Splenectomy was performed and thereafter recovery was uneventful. Histological examination of the spleen showed only hypertensive changes.

Right lumbodorsal sympathectomy was subsequently performed and followed by considerable symptomatic relief, although the blood-pressure remained high.

Two years after his first admission he returned suffering from vomiting and melæna, and died of renal failure ten days later.

At autopsy the peritoneal cavity contained a sero-sanguineous effusion. The alimentary tract was normal apart from a small ulcer at the midpoint of the small intestine. There were several green-stained dome-like elevations of varying sizes on the peritoneal surface of the gall-bladder. The largest of these was 8 mm. in diameter, and on section proved to be a small well-defined arterial aneurysm (*Fig.* 230). The liver was outwardly normal, but on section showed numerous small aneurysms, many of which were surrounded by small hæmorrhages and patches of necrosis. The heart was hypertrophied and dilated and the kidneys showed hypertensive changes.

Comment.—Severe hypertension was the main manifestation in this case of chronic polyarteritis nodosa. The only feature in the clinical course which might have suggested the diagnosis was the spontaneous rupture of the splenic artery. Platt (1948) has shown that severe hypertension in a young person is usually secondary and this case is no exception.

PATHOLOGY

The commonest malignant disease of the peritoneum is metastatic carcinoma. Peritoneal metastases are sometimes produced by sarcoma, malignant tumours of lymphoid tissue, malignant melanoma, malignant teratoma, and the embryonic tumours of childhood, especially neuroblastoma and nephroblastoma. Primary tumours of the peritoneum are very rare (Willis, 1948).

The metastases usually develop from cellular implantation across the peritoneal cavity, and the

ommonest source of such cells is the stomach, ovary, or large bowel. Occasionally the pancreas, iliary tract, kidney, or uterus prove to be the site f the primary. Carcinoma may also spread along ymph-vessels to abdominal lymph-nodes, infiltrate he peritoneum, and liberate malignant cells into the eritoneal cavity. Metastases may be blood-borne o the extraperitoneal tissues, to the liver, or to the owel submucosa, and extensive peritoneal involvement may develop in this way.

Gravity probably determines the distribution of free malignant cells in the peritoneal cavity. Cells which are not caught in peritoneal folds or recesses, such as those found along the attachment of the mesentery to the small intestine (*Fig.* 231), gravitate into the pelvic pouches or hernial sacs. Implantation occurs also in the great omentum, the epiploic appendices, or on the abdominal surface of the diaphragm.

The manner in which malignant cells implant themselves and grow on the peritoneum has been well described by Hertzler (1919) and by Sampson (1931). The cells provoke the peritoneum to form an exudate of fluid and fibrin and become adherent to and embedded in patches of fibrin attached to the peritoneum. The fibrin becomes organized by the growth into it of fibroblasts and capillaries, while invading mononuclear cells reach the free surface

FIG. 231.—A segment of small intestine, with its mesentery, obtained from autopsy on an old lady who had a carcinoma of the pelvic colon with widespread peritoneal metastases. The metastases are most numerous near the attachment of the mesentery to the intestine.

and become flattened and modified into cells indistinguishable from those which cover the adjacent peritoneum. The covering of these early implants with flat endothelial cells thus takes place simultaneously all over the surface of the nodule.

The fact that very young malignant implants are covered with apparently normal peritoneum has led some workers to believe, probably erroneously, that the main mode of origin of peritoneal implants is by lymphatic permeation (Carnett and Howell, 1932), and this emphasis on the extraperitoneal spread of metastases has been accepted by Bockus (1946).

The growth of peritoneal tumours is often accompanied by the development of clear straw-coloured, or blood-stained, ascites. Chylous ascites may develop if the thoracic duct becomes blocked by tumour deposits, while pseudochylous ascites may arise from the admixture of products of fatty degeneration with clear ascitic fluid (Willis, 1934).

MORPHOLOGY

Transcœlomic metastases are always raised above the surrounding peritoneum. Blood- or lymph-borne metastases appear first as flat patches lying

FIG. 232.—Multiple nodules of carcinoma on the peritoneum of the anterior abdominal wall of a woman suffering from carcinoma of the sigmoid colon. (*Natural size.*)

under the peritoneum, but if they continue to grow become raised and resemble those of transcœlomic origin.

The form taken by transcœlomic metastases is influenced by the condition of the peritoneum at the time of implantation of the tumour cells; the amount of fibrin and its arrangement; whether the tumour cells are enmeshed in the fibrin or lie on its surface; whether the fibrin becomes organized by fibroblasts alone or fibroblasts and vascular endothelium; the age of the fibrin; and the activity of the tumour cells.

A single primary may produce several different forms of metastasis. The form taken by the implant is not determined by the organ on which it is situated.

The main forms of malignant disease of the peritoneum are :—

1. *Discrete Nodules.*—This is the commonest form of peritoneal involvement (*Figs.* 231, 232). Nodules more than 0·5 cm. in diameter are opaque white, yellowish-white, grey, or reddish in colour and are often umbilicated. Nodules smaller than this are often semitranslucent and seldom umbilicated. Nodular development occurs when the implant grows at about the same rate in all directions.

2. *Plaque-like Masses.*—This is a relatively uncommon form. The plaques vary considerably in size and colour, and may or may not be covered with endothelium. They arise from the confluence of adjacent nodular deposits or when growth along the surface of the peritoneum exceeds growth in depth. Papillary adenocarcinoma of the ovary is the commonest cause of this form of metastasis.

3. *Diffuse Adhesive Forms.*—This form occurs at a late stage in the disease. The entire abdominal contents may be inextricably matted together and intestinal obstruction or fistula formation may occur. The adhesions are due to direct tumour infiltration

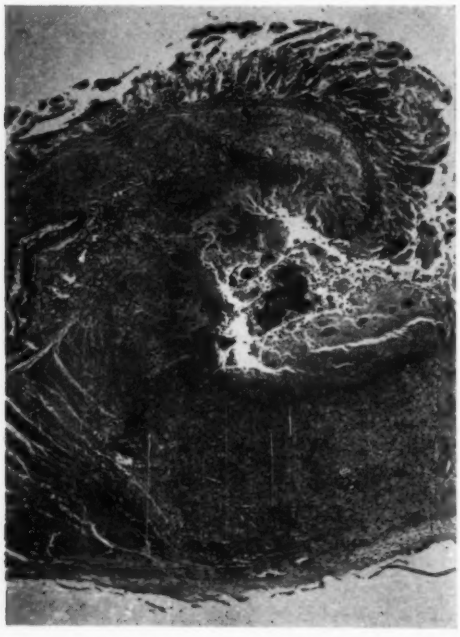

FIG. 233.—A deposit of reticulosarcoma cells in the submucosa of the ileum ulcerating through to the lumen and extending to the serosal surface, where it appeared as a flat, round, white patch. (× 18.)

from one organ to another, to confluence of adjacent implants, or to the organization of reactionary exudates.

4. *Flat Subperitoneal Forms.*—This is a relatively rare form. The implants are usually less than 0·5 cm. in diameter, round in outline, white in colour, and slightly translucent. They are due to blood-borne metastases developing in the submucosa of the intestines or the substance of viscera (*Fig.* 233).

5. *Cystic Forms.*—This is a rare form. The cysts may be sessile or pedunculated and vary greatly in size and thickness of wall. Small cysts are usually covered with endothelium. Cystadenoma of the ovary is the commonest primary in these cases. Rupture of an ovarian dermoid has been reported to produce cystic deposits of thyroid tissue (Morgen, 1924, cited by Willis, 1934).

6. *Pedunculated Forms.*—Pedunculated tumours vary considerably in size, length of pedicle, and colour. They are always covered with endothelium and they are usually due to papilliferous ovarian tumours.

7. *'Skin-graft' Form* (Sampson, 1931).—In this rare form of malignant disease of the peritoneum the only naked-eye change is loss of sheen. It is produced by the spread of a single layer of tumour cells over the surface of the peritoneum, often in palisade arrangement and without any embedding fibrous tissue.

8. *Pseudomyxoma Peritonei.*—In this rare form of malignant disease the peritoneum is lined and the abdomen usually distended with masses of pale or golden-yellow gelatinous material which may be loculated by fibrous septa. Pseudomucinous cystadenoma of the ovary is the usual primary; very rarely the condition is produced by some other mucin-producing tumour (Willis, 1948).

9. *Bizarre Forms.*—Rupture of a dermoid cyst or metastases from a teratoma may produce bizarre but often highly characteristic appearances. Tufts of hair may grow in the peritoneal cavity or the implants may produce masses of sebaceous material. These implants may lack genuine neoplastic qualities (Willis, 1934).

DIFFERENTIAL DIAGNOSIS

The following diseases and conditions may, on occasions, resemble malignant disease of the peritoneum.

1. Tuberculosis.—Discrete tubercles, common manifestations of tuberculous peritonitis, can closely simulate disseminated carcinomata. Early tubercles are greyish translucent granules, round, oval, or oblong in shape, 3–5 mm. in size, and firm in consistency (Baillie, 1794, cited by Hertzler, 1919). They quickly tend to undergo caseation and then appear as opaque, white or yellow, cheesy nodules (*Figs.* 223, 234).

Tuberculous granulation tissue in the retroperitoneal tissues (Edebohls, 1891), or on the surface

FIG. 234.—A portion of the mesentery of the small bowel of a 6½-year-old girl who died of tuberculous peritonitis. The mesentery is thickened, white, and opaque, and its surface is studded with numerous, small, round, white tubercles (four of which are indicated by arrows). The mesentery contains many enlarged caseating lymph-nodes which are not visible in this photograph. (× 0·9.)

of the peritoneum (Morris, 1914), may resemble plaque-like masses of malignant disease.

Dry or adhesive forms of tuberculous peritonitis, especially if accompanied by thick layers of granulation tissue, may resemble diffuse adhesive forms of malignant disease.

Acute miliary tuberculosis produces irregular greyish translucent lesions 1 or 2 mm. in diameter. These are usually seen on the surfaces of the liver and spleen, and may resemble flat subperitoneal nodules of growth.

Occasionally small, thin-walled, subserous cysts containing clear fluid (MacCallum, 1940) are found near tuberculous lesions of the intestine and have to be distinguished from cysts due to malignant disease.

Large thick-walled cysts may form where areas of tuberculous peritonitis are localized by adhesions. When these cysts occur in or near the female pelvis they may closely resemble malignant cystadenoma of the ovary (Morris, 1914).

Tubercles from 2 mm. to 2 cm. in diameter sometimes hang from the peritoneum by slender pedicles which may be up to 12 cm. long. These pedunculated tubercles closely simulate metastases from papillary cystadenoma of the ovary, even to their greyish or reddish colour. Diagnosis can be made only on histological examination (MacCallum, 1901).

Tuberculous peritonitis with mucus formation may on occasions simulate pseudomyxoma peritoneii (*see Case* 2).

When tuberculosis of the peritoneum is suspected, search should be made for a primary focus. If this is found and shows characteristic changes such as caseation or calcification, diagnosis is easy, but unfortunately the primary focus is sometimes indistinguishable from carcinoma and operative diagnosis may be impossible.

The presence or absence of ascites or enlargement of abdominal lymph-nodes is usually of no diagnostic value. Occasionally degenerating metastases in lymph-nodes simulate caseous tuberculosis. The great omentum, frequently involved in tuberculosis, can also be involved in malignant disease. In both cases it may be converted into a thick mass. The age of the patient is no guide to diagnosis.

Firm diagnosis of tuberculosis can usually be made on histological examination. Occasionally, however, there is such a profusion of endothelial cells and so little tubercle formation that the appearances suggest malignancy. If ascitic fluid is cultured tubercle bacilli can be found in about 50 per cent of tuberculous cases (Buchbinder, 1935).

2. Encapsulated Foreign Bodies.—Foreign bodies may enter the peritoneal cavity as the result of penetrating injuries or perforation of a viscus. Non-absorbable foreign bodies become encapsulated in fibrous tissue and subsequent laparotomy may reveal discrete nodules, plaque-like masses, pedunculated tumours, or diffuse tumours resembling those of malignant disease (*Cases* 3 and 6). Talc dusting powder introduced into the peritoneal cavity during the course of operations may set up a granulomatous reaction and present later as discrete nodules, plaques, or diffuse adhesions, indistinguishable to the naked eye from malignant disease (Borge and Refvem, 1950). Talc granulomata may appear as soon as seventeen days after the introduction of the talc (Mackey and Gibson, 1948), or may be delayed ten or even seventeen years (Roberts, 1947).

The diagnosis of encapsulated foreign bodies and talc granulomata should always be confirmed by histological examination. The polarizing microscope is of great value in these cases (*see Fig.* 229).

3. Lesions due to Chronic Sepsis and Mechanical Irritation.—Nodules or cysts indistinguishable to the naked eye from those of malignant disease may develop on peritoneum in the neighbourhood of chronic sepsis such as salpingitis, or on peritoneum subjected to prolonged mechanical irritation in a hernia. Similar nodules and cysts may also occur in association with malignant disease.

The nodules are usually 2–3 mm. in diameter, firm in consistency, grey or white in colour, and are sometimes translucent (*Case* 4, *Fig.* 225).

The cysts are usually a few millimetres in diameter, sessile, and translucent. Occasionally they may be as large as 1 cm. in diameter and pedunculated, especially when they occur at the fimbriated extremities of the Fallopian tubes (Williams, 1894).

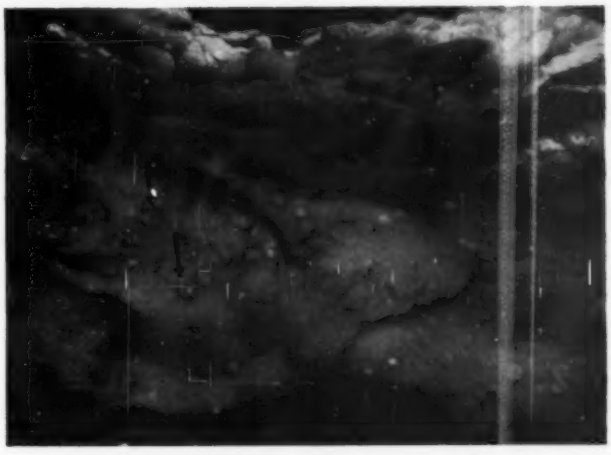

FIG. 235.—A portion of the great omentum of a woman, aged 69, who died six days after the onset of acute hæmorrhagic pancreatitis. The patches of fat necrosis lie level with the surrounding peritoneum and are chalky white in colour. Two of them, marked by arrows, are ringed with a narrow zone of inflammation. (× 0·75.)

The nodules and cysts are produced as the result of the deposition of flakes of fibrin. Nodules form when the fibrin becomes completely organized into fibrous tissue, and cysts when the fibrin is encapsulated by fibrous tissue and later absorbed and replaced by clear fluid. The nodules and cysts are rapidly covered with endothelium and the cysts lined with similar cells.

4. Fat Necrosis.—Fat necrosis, due to pancreatitis, trauma, or infection, may produce flat or nodular peritoneal lesions resembling those of malignant disease.

The flat patches are greyish or yellowish-white in colour, and can usually be distinguished from malignant deposits by their dense opacity (*Fig.* 235).

The nodules are due to fibrous encapsulation of cholesterol crystals released by fat necrosis, and examination under the polarizing microscope is usually necessary for their recognition.

5. Lesions due to Metazoan Parasites.—Hydatid disease may produce peritoneal lesions resembling those of malignant disease. So also may the *Schistosoma mansoni*, *Cysticercus pisiformis*, and *Tænia serratus* (Brumpt, 1936).

Hydatid disease occurs sporadically in the British Isles (Howell, 1939). The peritoneum becomes involved following the rupture of a primary hepatic cyst and this complication occurred in 16 per cent of the 1455 cases of hydatid disease recorded by

Barnett (1941). The shed scolices and brood capsules gravitate into the most dependent peritoneal pouches or are held in omental recesses, where they excite an inflammatory reaction and become embedded in granulation tissue and covered with apparently normal peritoneum (Dew, 1928). They may develop into large cysts and undergo fibrosis, caseation, or calcification. Small fibrotic nodules often coexist with large thick- or thin-walled cysts containing clear fluid. The primary cyst may persist in the liver, be marked only by a small scar, or become buried in adhesions. It should always be looked for if hydatid disease is suspected.

Hydatid disease is compatible with good health. Symptoms appear only when the cysts reach a large size. The diagnosis is very occasionally suggested by the history of an injury followed promptly by an acute abdominal syndrome and anaphylactic manifestations such as urticaria or pruritus. Usually, however, hydatid disease of the peritoneum is an unexpected operative finding. Diagnosis may be confirmed by biopsy, by the presence of eosinophilia, and in most cases by positive hydatid complement-fixation and Casoni intradermal tests.

6. Polyarteritis Nodosa.—Subperitoneal nodules are a common finding in polyarteritis nodosa and have to be distinguished from malignant nodules. They are formed by arterial aneurysms or the organization of periarterial hæmorrhages, and are usually greyish-yellow in colour and arranged along the course of visceral arteries, although they may appear anywhere (Grant, 1942).

Typically polyarteritis nodosa is a subacute pyrexial illness marked by severe general weakness and variegated visceral, neural, and cutaneous manifestations. Albuminuria is frequent and the disease may end in uræmia. Leucocytosis and anæmia are usually present (Davson, Bull, and Platt, 1948).

Abdominal manifestations often predominate, and appendicitis, cholecystitis, peritonitis, perforated peptic ulcer, or mesenteric thrombosis may be closely simulated. Intraperitoneal or intestinal hæmorrhage may occur (*Case* 7).

7. Gas Cysts (Pneumatosis) of the Intestines.—The rare condition of gas cysts could be mistaken for cysts of malignant disease. Gas cysts are thin-walled and translucent, and occur in clusters under the serosa, or in the mesenteries, of the intestines. They are usually accidental findings in malnourished cases of pyloric stenosis or carcinomatous intestinal obstruction (Dale and Pearse, 1950).

8. Enterogenous Cysts.—Enterogenous cysts, which are rare and usually accidental findings, must be distinguished from malignant cysts. They are found under the serosa, close to the bowel wall, contain mucin, and are lined by intestinal mucosa (Aird, 1949).

9. Splenosis.—Very rarely, traumatic rupture of a normal spleen is followed by widespread implantation and growth of splenic tissue on the peritoneum. The implants are dark bluish-red in colour, nodular, plaque-like, or pedunculated in form, and easily mistaken for malignant disease. Diagnosis is made on histological examination, which shows normal splenic pulp tissue covered with peritoneum (Bunch and Spivey, 1946).

10. Lesions due to Bacteria other than Tuberculosis.—Peritoneal nodules and adhesions which may be mistaken for malignant disease may be produced by many organisms, including *Mycobacterium lepræ*, *Pfeifferella mallei*, *Pasteurella pseudotuberculosis*, and *Brucella abortus* and *melitensis* (Lichtman, McDonald, Dixon, and Mann, 1946). Diagnosis may be suggested on clinical grounds and confirmed by biopsy, culture, or serological tests.

Abdominal actinomycosis may closely simulate malignant disease. Diagnosis may be difficult at laparotomy and depends upon biopsy and identification of *Actinomyces israeli* (Putman, Dockerty, and Waugh, 1950).

11. Ruptured Mucocele of the Appendix.—Rupture of a simple mucocele of the appendix may be followed by the development of pseudomyxoma peritoneii. In such cases the appendix shows a mucocele which may vary from 3 cm. in length to the size of a football. The gelatinous material is found free and implanted on the peritoneal surface, and although usually restricted to the neighbourhood of the appendix it may occasionally be found throughout the peritoneal cavity.

Pseudomyxoma peritoneii of appendicular origin has been reported in 15 male cases (Chaffee and Le Grand, 1942). It can be distinguished from mucoid metastases from a carcinoma of the alimentary tract by the absence of an alimentary primary, the absence of omental and visceral metastases, and the presence of free gelatinous material in the peritoneal cavity and a mucocele of the appendix.

Pseudomyxoma peritoneii of appendicular origin is more than three times commoner in women than in men, and in women has to be distinguished from pseudomyxoma peritoneii of ovarian origin. Distinction is usually made by finding an ovarian tumour and a normal appendix, although cases have been reported where a mucocele of the appendix and cystadenoma of the ovary occurred together (Voigt, 1937). In such cases the appendicular condition is usually secondary to the ovarian tumour.

DISCUSSION

Tubercle bacilli, foreign bodies, or parasites liberated into the peritoneal cavity are distributed and embedded in exactly the same way as malignant cells, and it is therefore not surprising that they may present almost identical appearances in their early stages. The true nature of a peritoneal implant will only become apparent when its specific component outgrows the non-specific, for example when tumour cells proliferate and form a large umbilicated mass or abundant and chaotically arranged new tissue.

When peritoneal lesions are discovered, search should always be made for other evidence which may aid diagnosis, such as a primary tumour, a tuberculous focus, an old peptic ulcer, or enlarged lymph-nodes suggestive of tumour of the lymphoid tissues (*Fig.* 236). Failure to find a primary tumour may be due to this being inpalpable (*Case* 1) or extra-abdominal.

Operative diagnosis should always be confirmed histologically. Histological diagnosis is not, however, always reliable (*Cases* 1 and 4). Metastases sometimes produce a very intense fibrous reaction

nd sections may then show either fibrous tissue lone (*see Fig.* 219) or isolated malignant cells so uried in fibrous tissue that they are difficult to lentify unless they can be demonstrated by special tains (*Fig.* 237).

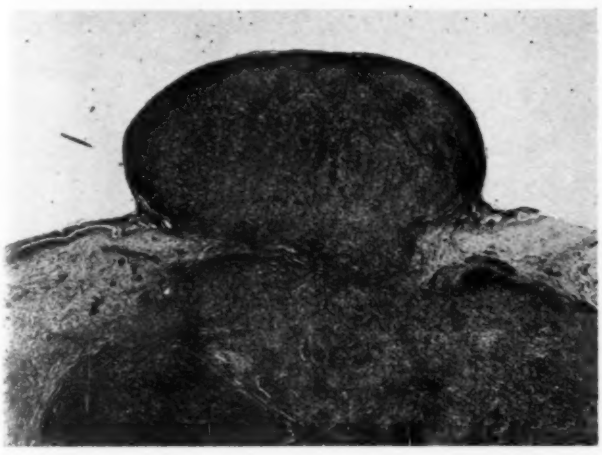

FIG. 236.—Peritoneal nodule found at autopsy on a 28-year-old man who died of Hodgkin's disease. High-power examination showed typical Hodgkin tissue. There was generalized lymph-node enlargement and involvement of the pleura and pericardium. (× 8.)

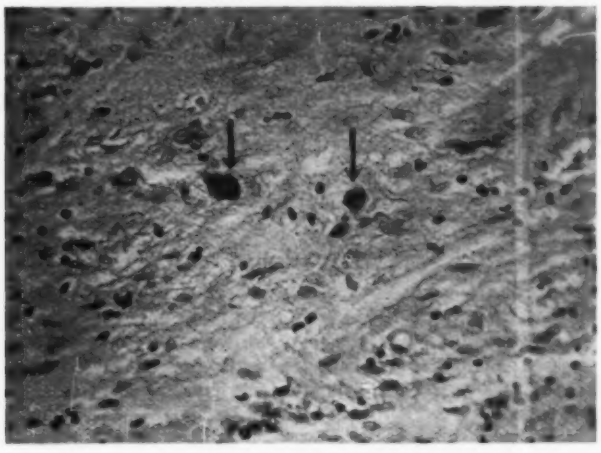

FIG. 237.—Peritoneal nodule from a case of mucus-secreting carcinoma of the stomach stained for mucus. Two cells, marked with arrows, contained large drops of mucus (signet-ring cells). These cells are buried in a mass of cellular fibrous tissue and were not detected on a section stained with hæmatoxylin and eosin. (× 480.)

Examination of the ascitic fluid found in malignant disease is said by Willis (1948) to show malignant cells in 50 per cent of cases. The interpretation of the cytological findings in ascitic fluid may, however, be very difficult (as in *Case* 2).

SUMMARY

Seven cases are reported illustrating some of the difficulties of diagnosing malignant disease of the peritoneum.

The commonest malignant disease of the peritoneum is secondary carcinoma, and this usually arises from the transcœlomic dissemination of an abdominal primary. Occasionally the peritoneum is involved by lymph- or blood-borne metastases from an extra-abdominal carcinoma, sarcoma, or other tumour. Primary tumour of the peritoneum is rare.

Malignant disease of the peritoneum appears in the following main forms: (1) discrete nodules, (2) plaque-like masses, (3) diffuse adhesive forms, (4) flat subperitoneal forms, (5) cystic forms, (6) pedunculated forms, (7) ' skin-graft ' forms, (8) pseudomyxoma peritoneii, (9) bizarre forms. Each of these may be closely simulated by the lesions of (*a*) tuberculosis, (*b*) encapsulated foreign bodies, (*c*) chronic sepsis, (*d*) mechanical irritation, (*e*) fat necrosis, and (*f*) various parasites and bacteria.

The only peritoneal appearances pathognomonic of malignancy are umbilicated nodules and large masses of chaotically arranged new tissue. Operative diagnosis, therefore, often depends upon associated findings such as an obvious primary tumour, a primary tuberculous focus, evidence of previous operation or of perforation of a viscus, or hydatid cysts in the liver.

The age of the patient, the presence or absence of ascites, tumefaction of the great omentum, or enlargement of the abdominal lymph-nodes are of little diagnostic value.

Histological examination should always be performed, even though this is occasionally inconclusive or even misleading. If histological diagnosis is difficult resort should always be made to the polarizing microscope and to mucicarmine-stained sections.

I am grateful to Mr. H. Burge, of the West London Hospital, for permission to publish *Case* 5; to Dr. L. W. Proger, Curator of the Museum of the Royal College of Surgeons, for permission to publish *Fig.* 231; to Mr. Norman Tanner and Dr. N. Orchard, of St. James Hospital, respectively, for permission to publish and for pathological reports on, *Cases* 3 and 6; and to the Staff of the Department of Pathology in the Postgraduate Medical School for all the other material.

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CEREBRAL ANGIOGRAPHY IN ABSCESS OF THE TEMPORAL LOBE

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THE purpose of this paper is to describe the part which cerebral angiography can play in the diagnosis and management of cases with abscess of the temporal lobe. The results of treatment of brain abscess have steadily improved in the last fifteen years (*Table I*),

Table I.—MORTALITY FROM BRAIN ABSCESS (*After Pennybacker*, 1950).

	Before 1938	1938–43	1943–49
Total number of cases	17	48	62
Mortality	94%	46%	29%

primarily as a consequence of advances in diagnostic and operative technique, assisted since 1943 by the use of penicillin and other antibiotics. The free use of these drugs has also introduced a new type of diagnostic problem, that of recognizing occult brain abscess in patients receiving systemic penicillin treatment for mastoiditis and infections elsewhere. In the investigation of these cases the most equivocal or minimal abnormal signs on neurological examination may be significant, and slight changes in the cerebrospinal fluid may be the only warning of the complication that has arisen. Plain radiographs may occasionally show pineal shift and, more rarely, gas in the cavity of the abscess; but more often than not little indication is given as to the presence or absence of such a lesion.

Other accessory methods of diagnosis have thus become increasingly necessary. In this clinic electroencephalography has been used in 17 cases of proved temporal lobe abscess, but it has provided evidence of a temporal lesion in only 11 of these patients and of its true nature in only 6. Air studies by ventriculography and encephalography are not always free from danger. This applies particularly to cases of temporal lobe abscess, probably because the expanded temporal lobe can exert pressure on the adjacent brain-stem more easily than can an abscess in any other part of the cerebral hemisphere. Furthermore, the ventriculograms may fail to provide the necessary information, for when there is much cerebral œdema the lateral ventricle on the side of the abscess may be very small and the foramen of Monro may be blocked; thus it may not be possible to get air to enter the ventricle on the affected side. In view of the shortcomings of these procedures a series of cases of temporal lobe abscess in which angiography has been employed has been studied, in order to ascertain the usefulness of the method and to find out what information as to the size and site of the lesion can be obtained.

CASE MATERIAL

In the last six years angiography has been performed at Oxford in 30 patients who were found to be suffering from cerebral abscess. In 12 cases the abscess was situated in the temporal lobe, and it is these which have been studied. In none was there any complication which could be attributed to the procedure, nor was there subsequently any deterioration of the patient's condition; indeed, several of them were critically ill at the time. In no case of temporal lobe abscess did the method fail to demonstrate the site of the lesion.

In 9 of these 12 cases the source of infection was a mastoiditis, one was bronchogenic (*Case* 2), one followed a streptococcal meningitis presumed to have arisen from an infected tooth socket (*Case* 9), and in the other (*Case* 12) the source is not known but was probably in the thorax. The youngest patient was aged 4 and the oldest aged 66 years.

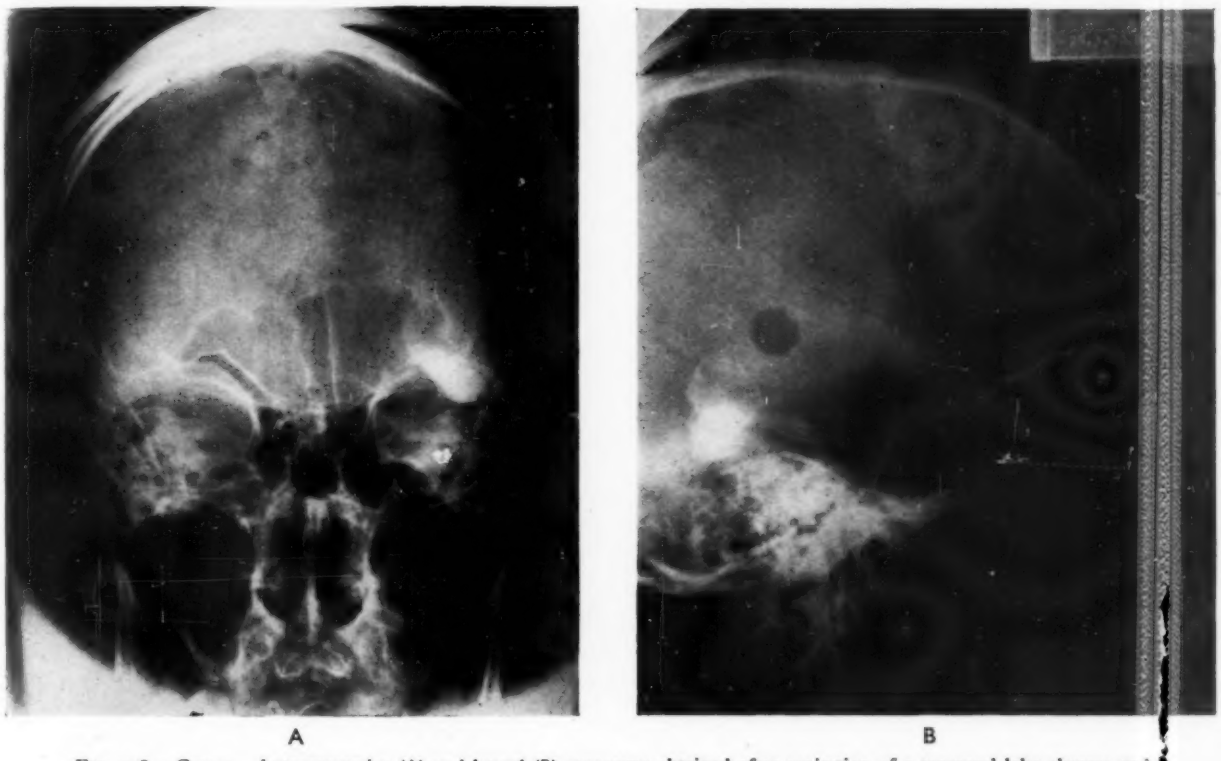
Most of the patients were first treated by aspiration of the abscess through a burr hole with instillation of penicillin (200,000 units) and thorotrast* (2 c.c.). Pyograms were then taken at intervals and further aspirations performed when indicated (*Fig.* 238). In 10 of the cases the abscess was subsequently removed when an adequate capsule had formed, that is to say, at least five or six weeks from the onset of symptoms. This was done by operation through a temporal bone-flap (Pennybacker, 1950).

In 9 cases angiography was first performed as a diagnostic measure before aspiration or removal of the abscess. In the other 3 it was first performed after aspiration and before excision, to provide further information about the lesion.

* Colloidal thorium dioxide.

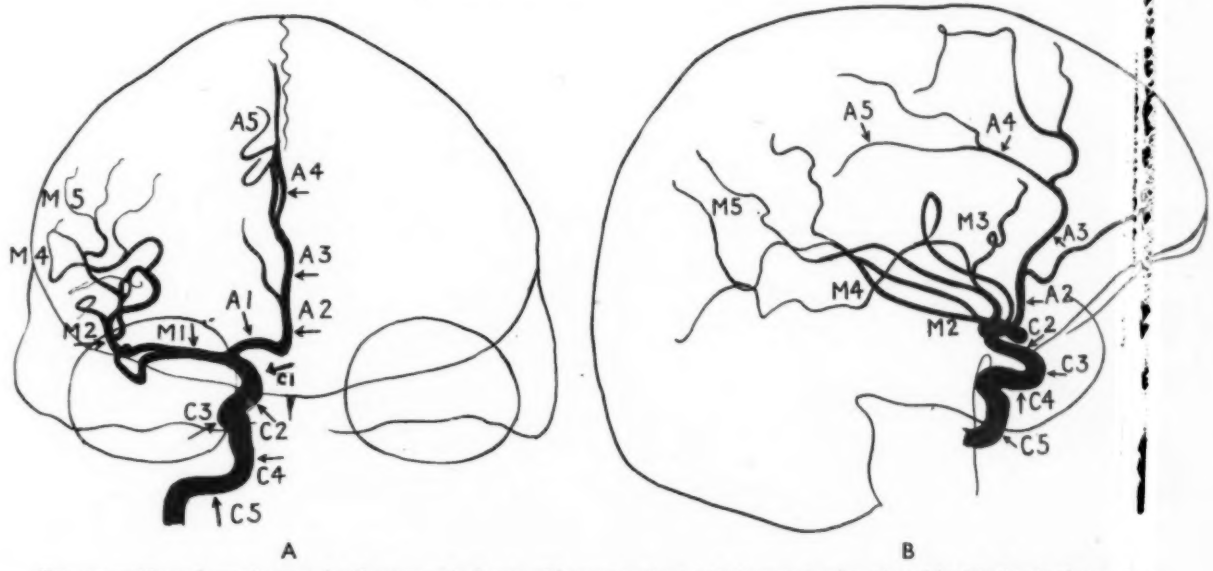
ANGIOGRAPHIC TECHNIQUE

The technique used for percutaneous angiography has been well described by Lindgren (1947), and that in use in this department has been given in detail by Curtis (1951). Briefly, it consists of percutaneous puncture of the common or internal carotid artery in the neck, under local anæsthesia. Diodone* (10–12 c.c. of 35 per cent solution) is the contrast medium used, and radiographs are taken in antero-posterior and lateral projections, 2, 4, and 8 seconds after the start of the injection. The technique is not difficult to acquire but needs practice. It is necessary to beware of the possibility of sensitivity to diodone, and this substance should also be avoided in patients with cardiac or renal disease.



A B

FIG. 238.—*Case* 9. Anteroposterior (A) and lateral (B) pyograms obtained after aspiration of a temporal lobe abscess and instillation of thorotrast.



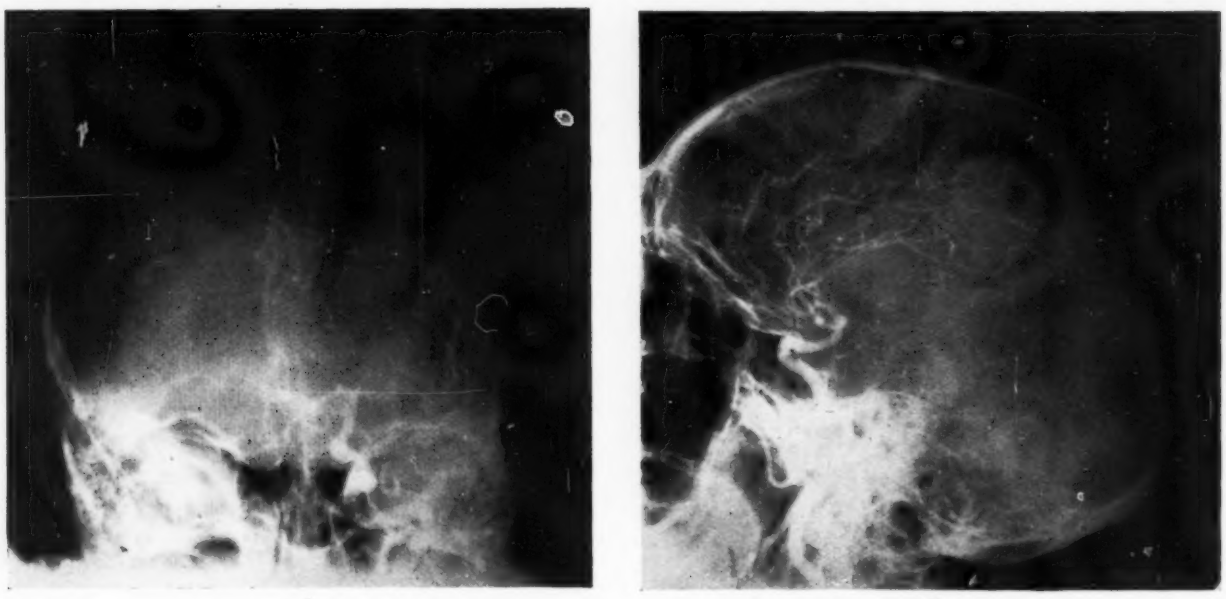
A B

FIG. 239.—Normal arteriograms in diagrammatic form. The nomenclature of the arteries introduced by Fischer is shown, but in this paper the need for its use has not arisen. A, Anteroposterior view. B, Lateral view. (*After Fischer*, 1938.)

* Pyelosil glaxo.

ANGIOGRAPHIC APPEARANCES

Fig. 239, modified from Fischer (1938), shows the normal arteriographic appearances in diagrammatic form. In space-occupying lesions within the cranium this vascular pattern is altered.



A B

FIG. 240.—*Case* 9. Arteriograms of a left temporal abscess showing the characteristic vascular displacements. A, Anteroposterior view. B, Lateral view.

ANTEROPOSTERIOR VIEW.—The features of a temporal lobe abscess in this projection are a shift of the anterior cerebral artery to the opposite side (*Fig.* 240 A), and in many cases a shift of the great vein of Galen in the same direction. The middle cerebral artery is usually elevated in its first part, especially when the abscess is anteriorly situated; it may not appear to be raised at all by those abscesses which are far back.

LATERAL VIEW.—This projection shows elevation of the middle cerebral group of vessels and an area in the temporal lobe may appear relatively avascular (*Fig.* 240 B). The normal distance of these vessels above the horizontal portion of the carotid syphon is taken to be 1 cm. (Engeset, 1944), and the degree of displacement is the increase in this distance. The shape of the distortion may give further information as to the precise location of the abscess in the middle fossa, the greatest displacement or stretching being above the abscess (*Figs.* 241 and 242).

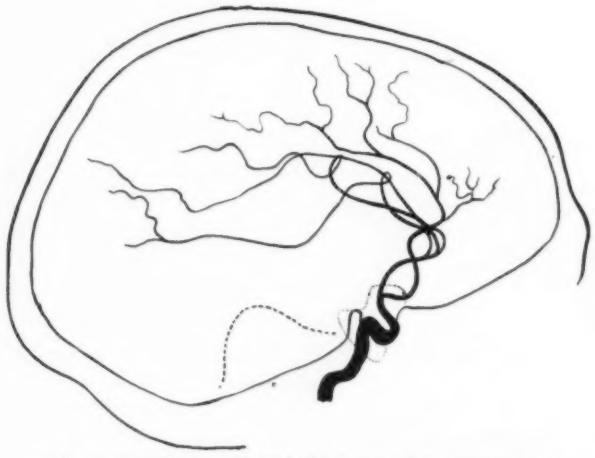


FIG. 241.—*Case* 8. Anteriorly situated right temporal lobe abscess. Lateral arteriogram to show the shape of the displacement. Only one loculus of the abscess is filled in the pyogram which has been superimposed. This loculus was adherent to the carotid artery and could not be removed with the rest of the abscess at operation. Seven weeks later it ruptured into the ventricle causing the patient's death.

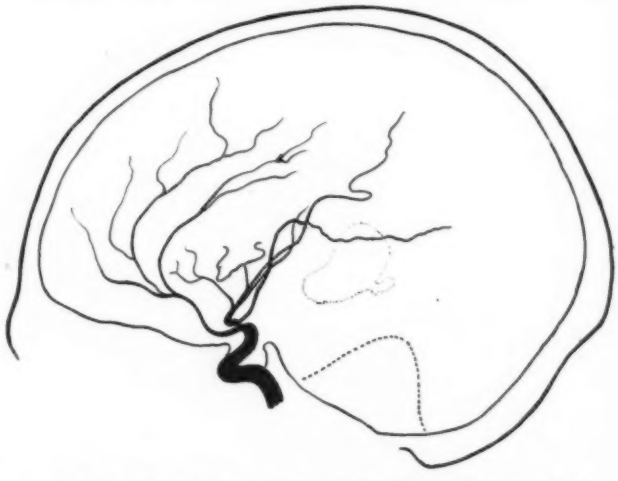


FIG. 242.—*Case* 12. Posteriorly situated left temporal lobe abscess. Lateral arteriogram to show the shape of the displacement, with pyogram superimposed. (Cf. *Fig.* 246.)

As stressed by Curtis (1951), accurate positioning of the patient is essential. In the lateral view, if the central ray is not directed just above, slightly anterior,

and parallel to the external auditory meati and at right angles to the film, the position of the cerebral vessels relative to each other, and to the skull, becomes altered, owing to parallax. In this manner slight tilting of the head or the beam in the lateral projection may produce apparent upward displacement of the middle cerebral artery, thus simulating the appearance of temporal abscess ; and, of course, if the tilt is the other way there is an apparent displacement of the artery in the opposite direction. Similar considerations apply to the anteroposterior projection.

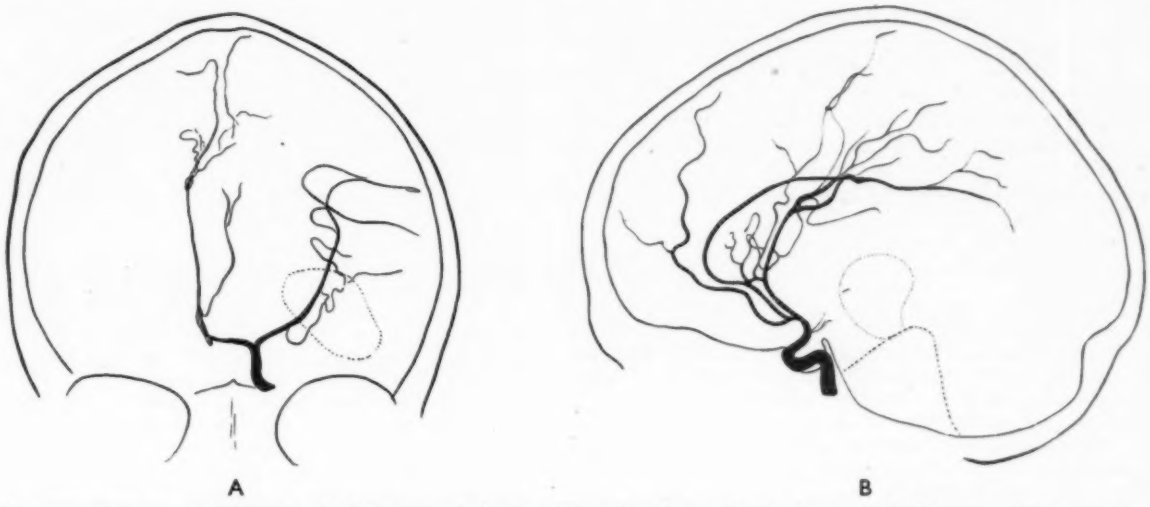


FIG. 243.—*Case* 10. Arteriograms of a left temporal abscess. There is only a moderate degree of displacement of the anterior cerebral vessels, but a gross elevation of the middle cerebral group is seen. The pyograms have been superimposed. A, Antero-posterior view. B, Lateral view.

ILLUSTRATIVE CASES

A temporal lobe abscess may present in one of four ways, and the following cases illustrate the use of angiography as an aid to the diagnosis of each group.

1. Intracranial Symptoms following Mastoid Infection.—In the first group there is an undoubted mastoid infection accompanied by headache or signs of raised intracranial pressure, with or without abnormal neurological findings to assist localization. The ear infection may be either acute or chronic.

Case 10.—A. A., aged 42 (R.I. 124168/50). Housewife.

This patient suffered from an attack of erythema nodosum in January, 1950, for which she received a course of sulphonamide treatment. A month later her left ear became painful and discharged sterile pus : she was then given systemic penicillin injections. On March 20 a left cortical mastoidectomy was performed, after which a thin discharge continued to come from the wound. A month after the operation she began to suffer from severe generalized headaches, so the mastoid cavity was explored again on April 24 but no locus of infection was found. Fluid blood was obtained on needling the lateral sinus. Pus continued to discharge from the wound, and she was admitted under our care on May 3 still suffering from very severe headache.

On examination she was alert and did not look very ill, but there was gross dysphasia of mixed type. There was a discharge from the mastoid wound which contained *Staph. aureus*, diphtheroids, and β-hæmolytic streptococci. The margins of the optic disks were slightly blurred, visual acuity could not be accurately measured on account of aphasia, and there was a complete right homonymous hemianopia. The left pupil was slightly smaller than the right. There was a mild right hemiparesis with hemianæsthesia, and an equivocal plantar response on the right side. Lumbar puncture just before admission had shown a cerebrospinal fluid (C.S.F.) pressure of 170 mm. with 120 mg. of protein per 100 c.c. and 2 cells per c.mm.

It was thought probable that this patient was suffering from a left temporal abscess, but the C.S.F. findings were not typical and an angiogram was performed to confirm the diagnosis. The films showed considerable upward displacement of the middle cerebral group with relatively little shift of the anterior cerebral vessels (*Fig.* 243). On the same day (May 5) a left temporal burr hole was made, the abscess was found at the first needling, and 20 c.c. of pus were aspirated from it. Penicillin (200,000 units) and thorotrast (2 c.c.) were introduced before withdrawing the needle. The pus was sterile on culture.

Following this aspiration her speech began to improve, the mastoid discharge lessened, and she said she felt better. The homonymous hemianopia fluctuated and at times seemed almost to have disappeared. However, on May 16 pyograms showed that the abscess shadow was increasing in size and a second aspiration was therefore performed (10 c.c. of pus, again sterile). Thereafter the aphasia continued to improve, so that four weeks after the first aspiration it was scarcely detectable. At this time the hemiparesis had cleared up except for a slight weakness of the right leg and the pyograms showed progressive diminution in the size of the abscess. In view of her considerable recovery, the fact that the pus from the abscess was sterile, and the fact that the lesion was obviously near the speech area, it was decided not to enucleate the abscess capsule unless she suffered a recrudescence of symptoms. This never happened, and she was discharged from hospital on June 8, at which time the lumbar C.S.F. pressure was 55 mm., and the fluid contained 45 mg. of protein per 100 c.c. and 14 cells per c.mm. She still had an incomplete right homonymous hemianopia.

When last seen in October she felt well and her visual fields were full (visual acuity, right, 6/5 ; left, 6/5). A slight degree of dysphasia was still detectable but the hemiparesis had virtually disappeared. She was beginning to do her housework, and three months later she reported by letter that she was doing full work and was free from symptoms. She had had no fits at any time.

In this case the clinical diagnosis did not present great difficulty, but the findings were unusual on account of the marked absence of drowsiness and normal C.S.F. pressure and cell-count. Angiography unequivocally settled the diagnosis, and, from the severity of the arterial displacement which it revealed (*Fig.* 243 B), indicated that surgical intervention was urgent despite the general well-being of the patient.

2. Raised Intracranial Pressure of Undetermined Origin.—In its second form the problem is that of a patient who has raised intracranial pressure, with or without localizing signs, and no known mastoid infection. A temporal lobe abscess may result from an apparently trivial middle-ear infection which subsides spontaneously or in response to treatment; or it may be metastatic from a lesion in the chest or elsewhere. Here angiography may provide the answer.

Case 2.—E. E., male, aged 66 (R.I. 64710/46). Retired grocer.

This patient was admitted on Nov. 8, 1946, with a history of a productive cough for a year with occasional small hæmoptyses, and frontal headaches for a period of five weeks. About the same time as the headaches came on he developed pain and tenderness in the left loin. A fortnight later he began to suffer some mental impairment with dysphasia which rapidly progressed. A week before admission an acute perinephric abscess containing *Staph. albus* was drained.

On admission he was co-operative and conscious though drowsy. He had gross dysphasia, both receptive and expressive. The fundi were normal but he had a right homonymous hemianopia. There was a right hemiparesis and hypo-algesia without increase of reflexes and with a flexor plantar response. Lumbar puncture showed a C.S.F. pressure of 165 mm. with a protein of 250 mg. per 100 c.c. and 300 cells per c.mm. His ears and nasal sinuses were normal. X-ray of the chest showed consolidation of the right upper lung field with a large cavity which was thought to be tuberculous. Plain skull radiographs showed the pineal shadow to be displaced to the right. The clinical diagnosis was that of a lesion of the left temporal lobe, probably an abscess, but possibly a tuberculoma or tumour.

An angiogram showed a huge left temporal lesion which appeared to be avascular and was thus probably an abscess or tuberculoma. An osteoplastic flap was turned without preliminary aspiration, and a large firmly-encapsulated abscess was removed. It occupied the greater part of the temporal lobe and also encroached on the frontal lobe across the Sylvian fissure.

The patient stood the operation well and the wound healed without incident, but there was little improvement in the function of the left hemisphere. He was discharged on Nov. 29, by which time he was able to sit out of bed. The final C.S.F. protein was 60 mg. per 100 c.c. with 8 cells per c.mm. and under normal pressure. This patient unfortunately died subsequently of pneumonia, the cerebral condition having given rise to no further symptoms. No post-mortem examination was performed.

Clinically this case appeared to be one of abscess of the left cerebral hemisphere, but the radiographs of the chest suggested pulmonary tuberculosis and raised the possibility of a tuberculoma. Angiograms did not distinguish between these two conditions, but did exclude a vascular tumour and also showed the precise situation of the lesion. It was thus possible to proceed immediately with an operation which was suitable for either of the lesions that might be encountered.

3. Cryptic Brain Abscess in Cases of Meningitis.—The third group consists of cases which present as meningitis but which also have an unsuspected abscess. As pointed out by Smith, Schiller, and Cairns (1946), this complicated situation is particularly common when the infection is due to anaerobic streptococci (5 cases out of 9 in their series).

Case 9.—A. D., male, aged 49 (R.I. 121254/50). Estate labourer.

This patient was perfectly well until Jan. 20, 1950, when he complained of headache and malaise. On Jan. 24 his face became swollen (probably from an infected tooth) and two days later he was confused and disorientated. He was admitted to another hospital and found to have 75,000 cells per c.mm. in the C.S.F., among which a few Gram-positive cocci were seen. He was given intrathecal and systemic penicillin and also sulphamezathine. He improved steadily and, according to his wife, became normal again apart from intermittent headache. The headache grew worse at the beginning of March and he was admitted to the Radcliffe Infirmary on March 7, as he had been found to have a high lumbar C.S.F. pressure (300 mm.) with 'raised protein' and 133 cells (80 per cent lymphocytes) per c.mm.

On examination he looked well and had neither fever nor headache. There were two very septic tooth stumps on the left side of his lower jaw. Little abnormality of speech was apparent in conversation but on testing he was found to have a profound nominal dysphasia, and almost complete inability to do simple mental arithmetic. His fundi showed severe bilateral papillœdema with exudates and hæmorrhages (V.A.R. J.4; V.A.L. J.6), and there was an attention defect in the right homonymous field. His hearing and ear-drums were normal. There was slight apraxia but no other evidence of motor or sensory disturbance apart from a minimal right lower facial weakness. Blood-pressure 150/90. Chest normal.

Angiography was performed on March 10 (*Fig.* 240). In the anteroposterior view the first part of the middle cerebral group was raised, and the anterior cerebral arteries and vein of Galen were moderately displaced to the right. The lateral view showed marked elevation of the middle cerebral group. It was concluded that there was a large avascular space-occupying lesion in the left temporal lobe which, when considered in conjunction with the history of a meningitis (? streptococcal), was thought to be an abscess. A temporal burr hole was made the next day and 10 c.c. of pus was aspirated. Penicillin (200,000 units) and thorotrast (2 c.c.) were put into the cavity and pyograms taken (*Fig.* 238). After aspiration the aphasia became worse, and he also had some transient impairment of sensation in the right limbs.

In the next two weeks an incomplete right homonymous hemianopia became apparent, and he had several amblyopic attacks. There were also slight signs of motor and sensory impairment on the right side, but his general condition showed gradual and definite improvement. On April 1 the capsule was considered to be firm enough for removal and the abscess was excised through an osteoplastic flap. Within the tissue removed were 3 loculi, in 1 of which was liquid pus; the other 2 contained granulation tissue.

This patient made slow but steady progress after operation. His aphasia improved, but owing to his slow recovery the possibility was considered that another loculus of the abscess might still remain. Angiography was therefore performed again, sixteen days after operation. There was still some displacement of the anterior and middle cerebral vessels, though less than before operation, and without any localized stretching. It was considered that there was no evidence of another abscess cavity, and his subsequent improvement justified this

onclusion. He made a good recovery and when last seen ight months after operation he was free from symptoms, lthough he had a complete right homonymous hemianpia, and a slight nominal dysphasia could still be demontrated. He was about to return to work.

Smith and her colleagues (1946) go so far as to tate that in streptococcal meningitis cryptic brain bscess is so common that ventriculography should lways be carried out, unless the patient is very well n himself and shows no trace of focal signs. From he experience which we have gained since that paper vas written there is no reasonable doubt that angiography is the better method to employ, unless the bscess is suspected to be in the cerebellum rather han in the cerebral hemispheres. In the case which has been described only one loculus of the abscess had been aspirated, so that the pyograms do not represent the full size of the lesion which was well shown in the arteriograms.

4. Cases of Protracted Mastoid Infection not doing well.—There remain those cases in which the patient is known to have a chronic mastoid infection which may or may not have been treated. He is not doing well and complains of headache and other vague symptoms, but has no objective neurological abnormalities and, in particular, no clinical signs of raised intracranial pressure. The lumbar C.S.F. pressure may be normal, but if the protein content of the fluid is raised, and particularly if there is also a mild pleocytosis, then an abscess may be suspected, and on occasion is eventually found. In such cases an angiogram may give valuable evidence of the presence or absence of intracerebral infection.

Case.—R. S., male, aged 22 (R.I. 30585/50). Welder. This patient was sent in as a possible intracranial abscess on Sept. 14, 1950. As an infant he had suffered from bilateral chronic otitis media and had had an intermittent discharge from the right ear until a Schwartze mastoidectomy was performed at the age of 9. After this he had an occasional discharge from the same ear until nine months before admission, when it became more profuse, purulent, and later sanguineous. Four months after this he experienced two attacks of vertigo with vomiting. On the second occasion he had diplopia on looking to the left, and this was followed by severe frontal headache and pain in the right temporal region. He then began to improve and the diplopia disappeared, but anxiety was occasioned by finding a C.S.F. with a protein content of 70 mg. per 100 c.c. and 13 lymphocytes per c.mm.

On admission there was a purulent discharge from the right ear with a polyp perforating the drum. There was no useful hearing in this ear and the Weber test was referred to the opposite side. There was no labyrinthine function on the right. The remaining cranial nerves were normal, and there was no ataxy or weakness and no unsteadiness of gait. The lumbar C.S.F. pressure was 130 mm. with a protein of 60 mg. per 100 c.c. and 28 white cells per c.mm., 26 of which were lymphocytes; it was sterile on culture. The plain radiographs showed a large mastoid cavity, with no evidence of cholesteatoma, and a central calcified pineal body. Angiography was performed on the right side and the appearances were normal, that is to say there was no displacement of the middle cerebral vessels as in temporal lobe abscess, and no evidence of hydrocephalus due to a cerebellar abscess. Four months later this patient reported himself free from headache and giddiness.

This case was considered to be one of right-sided labyrinthitis and chronic suppurative otitis media with aural polypi and no evidence of intracerebral complications, and the subsequent course confirmed that nothing more serious was the matter. However, there was undoubtedly some meningeal infection, and without angiography it would have been difficult to be certain that the infection was from the labyrinth and not from an unsuspected brain abscess. Encephalography could also have decided the issue, but not without some risk of lighting up the labyrinthine-meningeal infection.

DIFFERENTIAL DIAGNOSIS

It is now necessary to review systematically the part which angiography may play in the differential diagnosis of temporal abscess. There are other intracranial complications of infection of the petrous bone which clinically simulate an abscess of the temporal lobe. These have to be considered, and also cases of non-infective lesions of the temporal lobe, such as tumours, in patients who happen to have also a chronic ear infection.

As yet we have little angiographic information about the lesions in the infective group, because, apart from cerebellar abscess, they are relatively rare. The neurosurgical problem is usually that of excluding the presence of a collection of pus. As experience grows it might be expected that angiography could be made to depict small collections of pus over the surface of the brain, either superficial or deep to the dura, but it is probable that its chief value in these cases will remain that of excluding abscess formation within the substance of the brain.

Extradural and Subdural Abscess, Petrositis.—We have no angiographic experience of the first of these lesions, which may sometimes be distinguished clinically from temporal lobe abscess by the severity of the local pain. Subdural abscesses in the common situation on the medial surface of the cerebral hemisphere may show a depression of the anterior cerebral artery, as described by Schiller, Cairns, and Russell (1948); and also, in the anteroposterior view, a curved displacement of the medial surface of the hemisphere away from the middle line with its convexity towards the side of the lesion. A normal angiogram without displacement of the middle cerebral group would be expected in petrositis.

Cerebral Thrombophlebitis.—In these cases there is sometimes a slight lateral displacement of the anterior cerebral vessels towards the opposite side, on account of œdema of the affected hemisphere, but the absence of upward displacement of the middle cerebral group again provides the evidence for exclusion of a temporal lobe abscess.

Sinus Thrombosis.—This condition may be associated with temporal lobe or cerebellar abscess. What the frequency of this association is at the present time is uncertain, but in 1931 Evans found that in a series of 109 cases of brain abscess, the infection had spread from the lateral sinus in 37. In 28 of these the abscess was in the cerebellum, and in 9 it was in the cerebrum. Angiography is of considerable value in excluding temporal lobe abscess in these cases, as in the following example.

Case.—J.J., male, aged 10 (R.I. 127980/50).

This boy, who was admitted on July 17, 1950, had a left cortical mastoidectomy performed for an acute on chronic mastoid infection in April, 1948. He did well

after this operation but there was an intermittent discharge of pus from his left ear during the next two years. In July, 1950, the pain in the left ear returned, and two weeks later he had a generalized epileptic fit. The next day he was confused and he was found to have papillœdema and a lumbar C.S.F. pressure of over 300 mm. with 10 mg. of protein per 100 c.c. and 5 cells per c.mm. On July 13 the left mastoid was reopened and foul subperiosteal pus was found; a hæmolytic streptococcus—Group B—was

FIG. 244.—Second phlebogram (8 sec.) showing the confluence of the sinuses. Anteroposterior view. (Normal angiogram.)

grown on culture. The sigmoid sinus was exposed at this operation, and its wall was found to be grossly thickened, but fluid blood could be aspirated without difficulty. His condition improved for a short time after operation but then deteriorated with return of slight confusion.

He was transferred to this department where he was found to have 2D papillœdema in the right eye (V.A.R. J.4) and 1–2D in the left (V.A.L. J.16); vision in the left eye was poor and associated with a squint which had been present from infancy. The visual fields were full. There was bilateral kernigism and neck stiffness but there were no lateralizing signs. Lumbar puncture showed a pressure of over 500 mm. with a brisk rise and fall on compression of the right jugular vein, but no response on compression of the left. The C.S.F. protein was now 35 mg. per 100 c.c. with no cells, and the fluid was sterile on culture. His alert clinical state in the presence of papillœdema and raised intracranial pressure, and the normal protein and absence of cells in the C.S.F., with failure of jugular response on the left side, suggested a diagnosis of otitic hydrocephalus.

Angiography was performed, and the presence of an abscess was excluded since the arteriogram showed no vascular displacement or stretching. No further information could be deduced on the question of jugular or sinus thrombosis, and a sinogram performed later by injecting diodone into the superior sagittal sinus did not give any more help.

He was discharged on August 4, with no cells in the C.S.F. and a protein of under 10 mg. per 100 c.c., the pressure being 250 mm. He had returned to his normal mental state and the neck rigidity was gone. When seen in October, his disks were flat but with filling of the cups and some secondary optic atrophy (V.A.R. J.2; V.A.L. J.18); there was still some aural discharge but in other respects he had returned to normal.

In this case the clinical picture suggested an otitic hydrocephalus following thrombosis and obstruction of the left lateral sinus, but the intermittent mental confusion made it necessary to exclude an abscess by means of angiography. Angiography of the lateral sinuses has so far proved disappointing because of the inconstancy of sinus shadows in the normal. Good filling of them (*Fig.* 244) is not very common. Direct injection of the contrast medium into the sinuses may, with further experience, prove of value, and a recent paper by Ray, Dunbar, and Dotter (1951) describes a technique by means of which good results have been obtained.

Cerebellar Abscess.—In this condition there is no displacement of the carotid system, except that which is due to hydrocephalus—a stretching of both middle and anterior cerebral groups caused by the enlarged lateral ventricles.

Abscess of Other Parts of the Cerebral Hemispheres.—According to the situation of the abscess, various displacements of the vascular tree may be produced. None of these will elevate the middle cerebral vessels and confusion with a temporal lesion is unlikely.

Localized Hydrocephalus of the Inferior Horn of the Lateral Ventricle.—Cairns, Daniel, Johnson, and Northcroft (1947) have described an unusual condition which may be mistaken clinically and angiographically for an abscess of the temporal lobe. Following gunshot wound or abscess of the parietal lobe, or ventricular hæmorrhage, the roof and the floor of the central part of the lateral ventricle may become adherent, with the result that the inferior horn of the ventricle is closed off and becomes progressively dilated by C.S.F. produced within it.

Case.—G. S., male, aged 22.

This was a patient in whom the above condition followed a fronto-parietal abscess, which had in turn followed a temporal lobe abscess that had been removed by operation through a lateral osteoplastic flap. *Fig.* 245, A, shows the angiogram on which the diagnosis of fronto-parietal abscess was made. This abscess was treated by needling, but the patient failed to make progress. In the angiogram (*Fig.* 245, B) taken 4 months after the previous films, the displacement of the middle cerebral group is reversed. The appearances are indistinguishable from those of temporal lobe abscess. The condition of localized hydrocephalus of the inferior horn was found at operation, and was successfully treated by destruction of the choroid plexus of this part of the lateral ventricle.

Neoplasm.—Angiographically brain abscess usually appears as an avascular lesion. Wickbom (1948) has described an angiogram in which capillary vessels were seen around an abscess cavity, but it was a very chronic lesion and this unusual finding probably only occurs with abscesses of long standing. (*See also* Lima, 1950.) In most tumours of the brain angiography reveals some part of the tumour circulation. Angiomas, meningiomas, glioblastomas, and some other neoplasms, often show widespread evidence of their abnormal vasculature, and there is

usually at least a fringe of pathological circulation. There is no difficulty in distinguishing these tumours angiographically from abscess. However, in cystic or avascular neoplasms, particularly astrocytomas, no tumour circulation may be seen ; in the temporal lobe the displacement of the main cerebral vessels may be identical with that produced by abscess. In these cases confusion should seldom arise if all the details of clinical examination and history are taken into account, and, furthermore, re-examination of angiograms in avascular tumour cases will sometimes show a small area of pathological circulation.

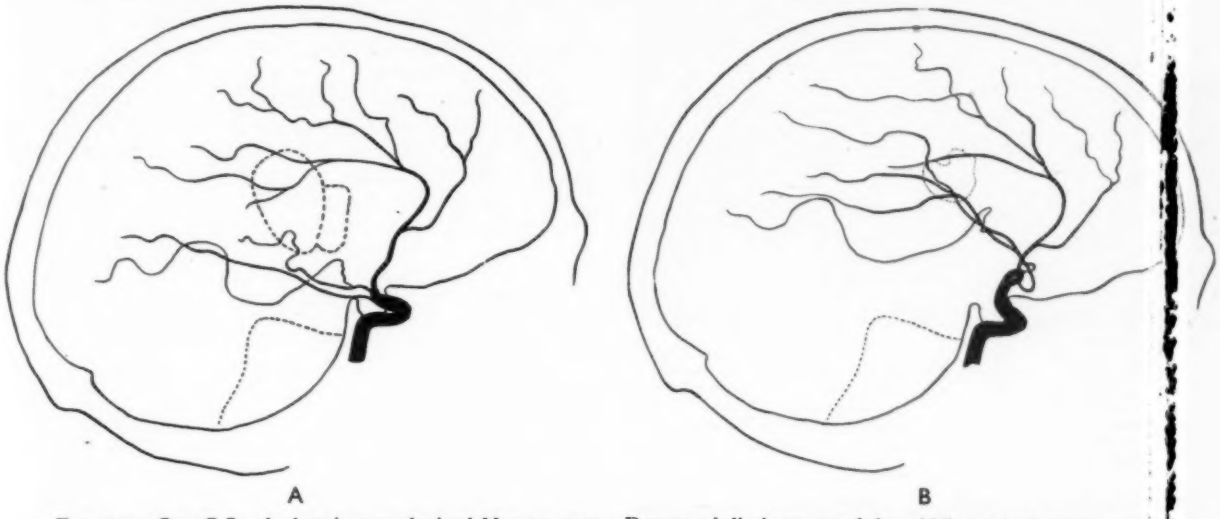


FIG. 245.—*Case* G.S. A, Arteriogram obtained May 24, 1950. Downward displacement of the middle cerebral group caused by a right frontoparietal abscess. B, Arteriogram obtained Sept. 27, 1950, after treatment of the abscess by aspiration, showing shrinkage of the abscess cavity and elevation of the middle cerebral vessels caused by localized hydrocephalus of the inferior horn.

REPEATED ANGIOGRAPHY

Percutaneous angiography can be repeated after an interval of a few days without causing harm.* After removal of an abscess the angiographic picture returns to normal and *Case* 12 was re-examined four months after operation to illustrate this ; it will be realized, however, that the time taken for it to happen varies from patient to patient. The lateral angiogram is shown in *Fig.* 246, and it can be seen by comparison with *Fig.* 242 that the previous distortion has disappeared. In a recent paper Fabritius, Frøvig, and Kristiansen (1949) have described the use of angiography as a means of following the progress of an abscess in a case in which the lesion was not removed. The abscess had been tapped at mastoidectomy and then left to resolve on its own with the aid of penicillin treatment. Angiograms taken at monthly intervals showed the vascular pattern to have returned to normal after two months. This application of the method is of particular importance when thorotrast pyograms are not used, as is the custom in some clinics (Taylor, 1950).

Angiography may also be of assistance after operation for excision of an abscess, when the clinical state leads to the suspicion that the abscess has not been completely removed, or that another has formed (cf. *Case* 9).

* This was recently done 5 times in the course of ten weeks in a patient with a subdural hæmatoma.

RELATION OF ANGIOGRAPHIC APPEARANCES TO THE PATHOLOGICAL CONDITION

The phases of development of a cerebral abscess in man have been described by Carmichael, Kershman, and Adson (1939). At a stage when they are demonstrable by angiography there is a central zone of necrosis and pus formation, outside which is a layer of hyperæmic granulation tissue containing fibroblasts. In the brain beyond this, and for some distance farther, there is a varying amount of gliosis (proliferation of astrocytes) and œdema (*Fig.* 247). This œdema is space-occupying on its own account. The capsule is only composed of loosely bound fibroblasts, reinforced by intertwining vessels and covered by a thin lamina of compressed brain substance showing gliosis. This layer confines the septic process and is not firmly established for about

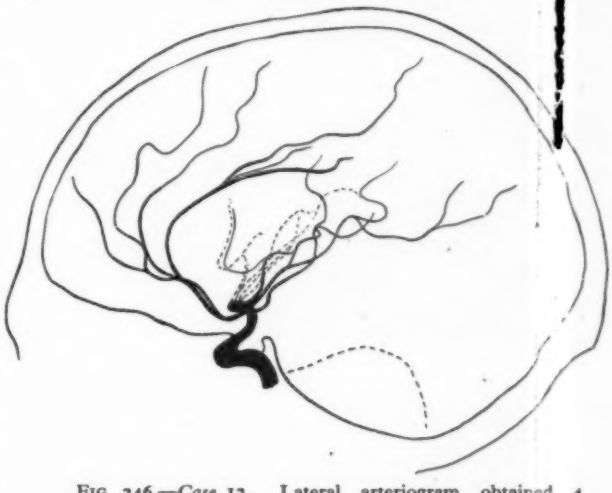


FIG. 246.—*Case* 12. Lateral arteriogram obtained 4 months after removal of a left temporal abscess. The interrupted lines show the course of the middle cerebral group of arteries before operation. The position of these vessels has returned to normal.

three weeks. At operation a plane of cleavage is found around it. Where the capsule is less marked, and this is usually where it lies deep in the white matter or adjacent to the ventricle, the abscess may

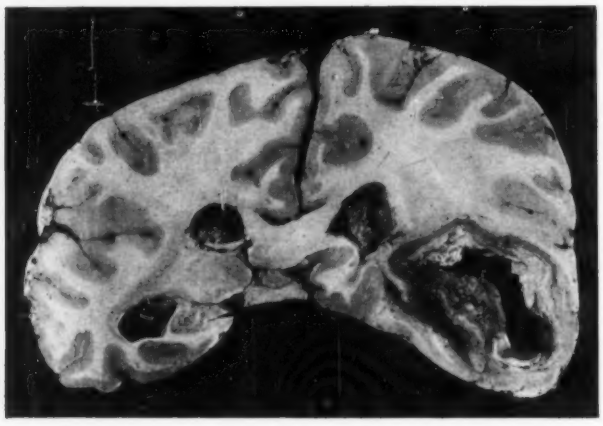
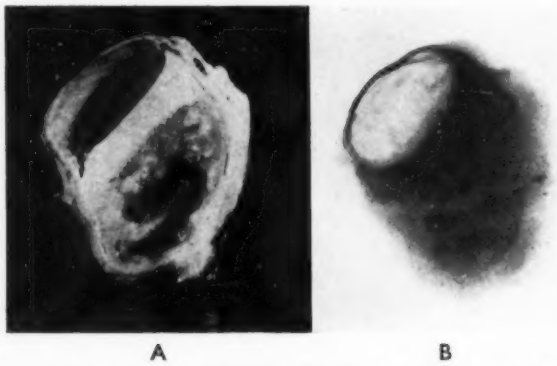


FIG. 247.—Abscess of the right temporal lobe treated by aspiration on one occasion only. An area of necrotic brain tissue is seen below the cavity and adjacent to the inferior horn of the lateral ventricle, which is almost completely occluded. The right hemisphere is larger than the left, and the œdematous white matter occupies a greater area than does the normal white matter on the other side.

form diverticuli or loculi. In temporal lobe abscesses, as in those elsewhere in the cerebral hemispheres, approximately 50 per cent are multilocular (32 cases).

We can gain some indication of the significance of the angiographic displacements by comparing them with the appearances of thorotrast pyograms of the abscess cavity. When thorotrast has been



A B

FIG. 248.—A, Operation specimen of a temporal lobe abscess, cut longitudinally to show the thickness of the capsule. The pus has formed layers of varying thickness, above which is a bubble of air. B, Radiograph of the same specimen to show the depth to which the thorotrast has penetrated in macroscopic amounts. Some of the contrast has been left in the track of the aspirating needle. (× ⅔).

injected into an abscess most of it remains in the pus, but some of the granules are taken up by macrophages in the capsule, so that a layer of these cells filled with thorotrast can be seen lining the cavity and scattered in the adjacent tissues (Falconer, McFarlan, and Russell, 1943). Macroscopic amounts of thorotrast only penetrate a short distance into the capsule, as can be seen in *Fig.* 248, and this is the outline of contrast in a pyogram. When the abscess is multilocular, thorotrast may not have been introduced into all the cavities (Tutton and Shepherd, 1949).

It can be seen, therefore, that pyograms frequently do not show the true size of an abscess, and they never show the extent of the œdema which, by raising intracranial pressure, may endanger the patient's life more than does the abscess itself. The displacement of vessels seen in angiograms, on the other hand, is caused not only by the volume of the abscess and its capsule but also by the surrounding œdema. The vascular distortion thus indicates the total lesion and may be fairly considerable even though the abscess cavity itself is small, as in *Case* 3. This is not, however, intended to imply that the practice of injecting thorotrast after aspiration of an abscess should be discontinued, as much information is to be gained from its use, and there is some evidence that it may stimulate capsule formation.

In this study an attempt is made to correlate the degree of vascular displacement, the measurement of

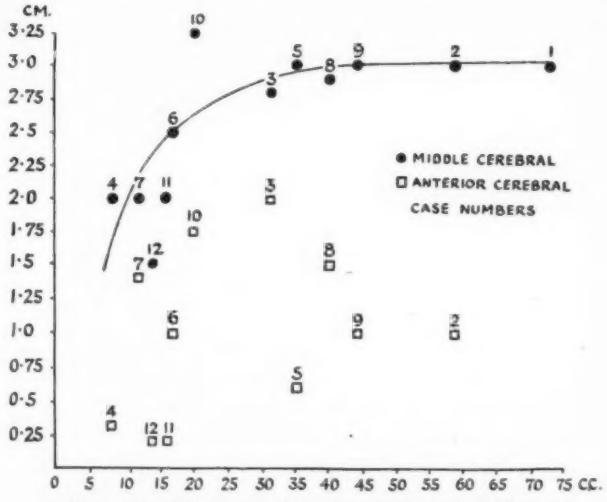


FIG. 249.—Displacements of the anterior and middle cerebral vessels compared with the estimated volume of the lesion in 12 cases of temporal lobe abscess.

which has been described, with the approximate size of the abscess, estimated in this series by one of the following methods: (1) The operation specimen was measured in those 5 cases in which angiography was performed shortly before excision of the abscess and when the lesion was in a more or less static condition. (2) When angiography was performed immediately before aspiration of an abscess, the volume was estimated from the amount of pus aspirated and the size of the shadow in the pyogram, assisted where possible by the operation specimen obtained at a later date (6 cases). (3) In *Case* 4 the intervals between aspiration and angiography, and between angiography and operation, were both too long to be discounted, and an approximate figure was taken from three volumes on different dates.

The volume of each lesion was estimated by means of a plasticine model made from the data available. These figures, together with the vascular displacements in the corresponding angiograms, are summarized in *Table II.*

The estimated volume is now plotted against the shift of the middle cerebral vessels, and it will be seen that they are roughly proportional to one another (*Fig.* 249). On the other hand there is no

relationship between this volume and the displacement of the anterior cerebral vessels; there is also no relationship to displacements of the great vein of Galen. The amount of displacement and the course of the middle line vessels (the anterior cerebral arteries and vein of Galen) depends to some extent on the position of the lesion in the temporal lobe, but this alone does not account for the variable shift found in this series. The conclusion to be drawn, therefore, is that the degree of elevation of the middle cerebral group gives an indication of the size of the lesion, though not necessarily of the size of the abscess alone. The shift of the middle line structures, on the other hand, does not allow any inference as to the volume of the lesion or the urgency with which it should be treated.

Table II.—Abscess Volume and Vascular Displacement

Case	Method of Estimating Volume (*See* Text)	Volume at Time of Angiography	Shift of Anterior Cerebral	Shift of Middle Cerebral	Shift of Vein of Galen
1	1	73 c.c.	No A.P. View	3·0 cm.	No A.P. View
2	1	59 c.c.	1·0 cm.	3·0 cm.	Not visible
3	2	31 c.c.	2·0 cm.	2·8 cm.	0·5 cm.
4	3	7 c.c.	0·3 cm.	2·0 cm.	0·3 cm.
5	1	35 c.c.	0·6 cm.	3·0 cm.	1·0 cm.
6	2	17 c.c.	1·0 cm.	2·5 cm.	Not visible
7	1	12 c.c.	1·3 cm.	2·0 cm.	0·5 cm.
8	2	40+ c.c.	1·5 cm.	2·8 cm.	Not visible
9	2	44 c.c.	1·0 cm.	3·0 cm.	0·5 cm.
10	2	20+ c.c.	1·75 cm.	3·25 cm.	1·5 cm.
11	2	16+ c.c.	0·2 cm.	2·0 cm.	Central
12	1	14 c.c.	0·2 cm.	1·5 cm.	Central

SUMMARY

1. The place of angiography in the diagnosis and management of temporal lobe abscess is discussed, with a description of the angiograms obtained. A study has been made of 12 cases examined by this method.

2. Angiography has been found to be a reliable and safe aid in the diagnosis of abscess of the temporal lobe, and subsequently as a means of demonstrating certain complications and observing progress. Cases illustrating these uses have been described. In this series there have been no complications attributable to the procedure.

3. The relation of the vascular displacements to the lesion is considered. Displacements of the middle cerebral group are shown to be roughly proportional to the total volume of the abscess.

I wish to express my sincere thanks to Professor Sir Hugh Cairns for the care and patience with which he has helped and advised in the preparation of this paper; also to Mr. Joe Pennybacker, whose assistance and criticism were most valuable. This department is indebted to those surgeons who treated the patients before they were referred to us, particularly to Mr. N. E. Kendall of the Leicester Royal Infirmary. Miss A. J. Arnott reproduced the tracings for which I am very grateful.

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TROCHANTERIC FRACTURES OF THE FEMUR

By W. G. FRANCE

ORTHOPÆDIC TUTOR AND SENIOR REGISTRAR, THE GENERAL INFIRMARY AT LEEDS

AND C. R. BERKIN

SENIOR REGISTRAR, THE GENERAL INFIRMARY AT LEEDS

DURING the last decade, a considerable number of papers from American surgeons have extolled the advantages of internal fixation in the treatment of trochanteric fractures. By contrast, British opinion has been slower to depart from conservative measures. Thornton (Thornton and Sanderson, 1937) was the first to devise an internal splint, consisting of a plate, one end of which fitted over the head of a Smith-Petersen nail and fixed to it by means of a screw. Since then, at least ten varieties of nail-plate have been described, some cast in one piece and others consisting of nail and detachable plate.

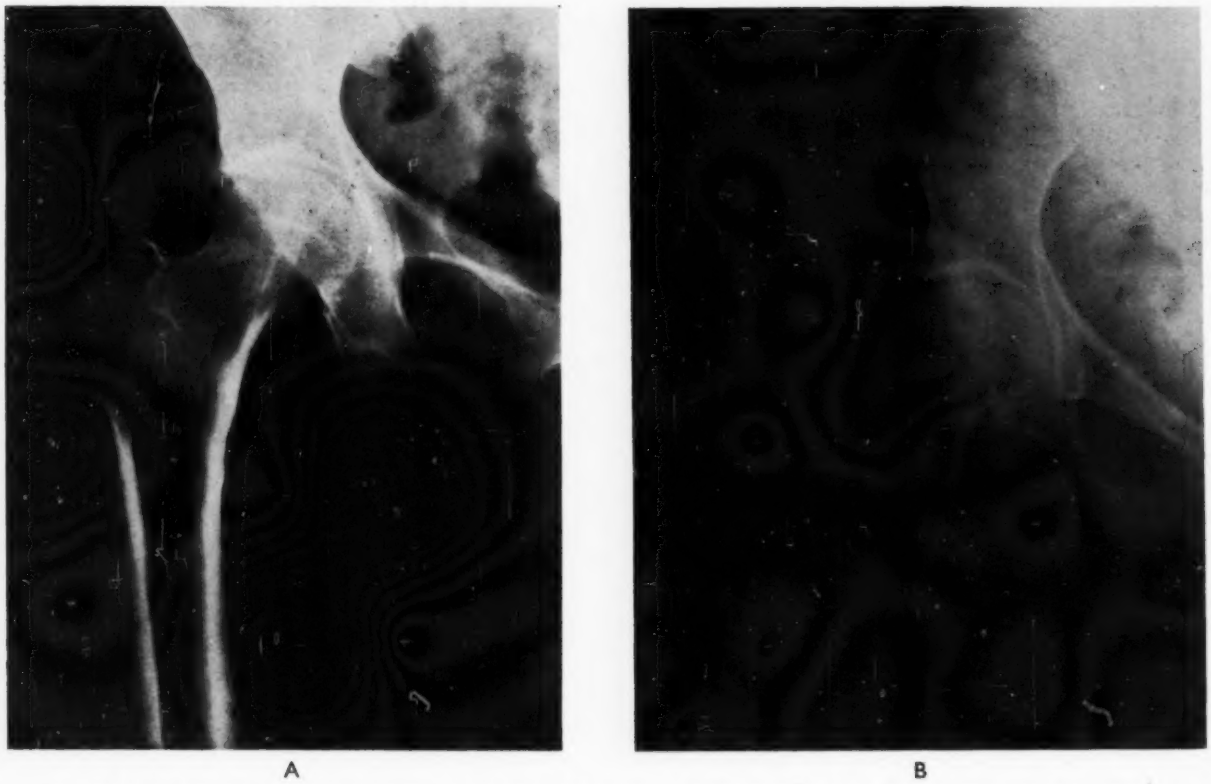
This report deals with 82 consecutive trochanteric fractures treated at the Leeds General Infirmary during the period January, 1946, to October, 1949, inclusive. A comparison is made of some of the results of treatment by internal fixation and conservative methods.

Sex.—Females (62 cases—78 per cent) were more commonly affected than males (20 cases).

Age.—The average age was 69·5 years, the youngest patient being 21 years and the oldest 93 years; 56 per cent of the patients were over the age of 70.

Side.—The left femur was fractured in 48 cases (58·5 per cent).

Associated Conditions.—As one would expect, associated pathologies were frequent. Fourteen patients presented the following conditions: Parkinsonism, 3 cases; hemiplegia (long-standing), 2; chronic bronchitis and emphysema, 2; pernicious anæmia, 2; fractured pelvis, 2; hypertension, 1; auricular fibrillation, 1; multiple gummata, 1.



A B

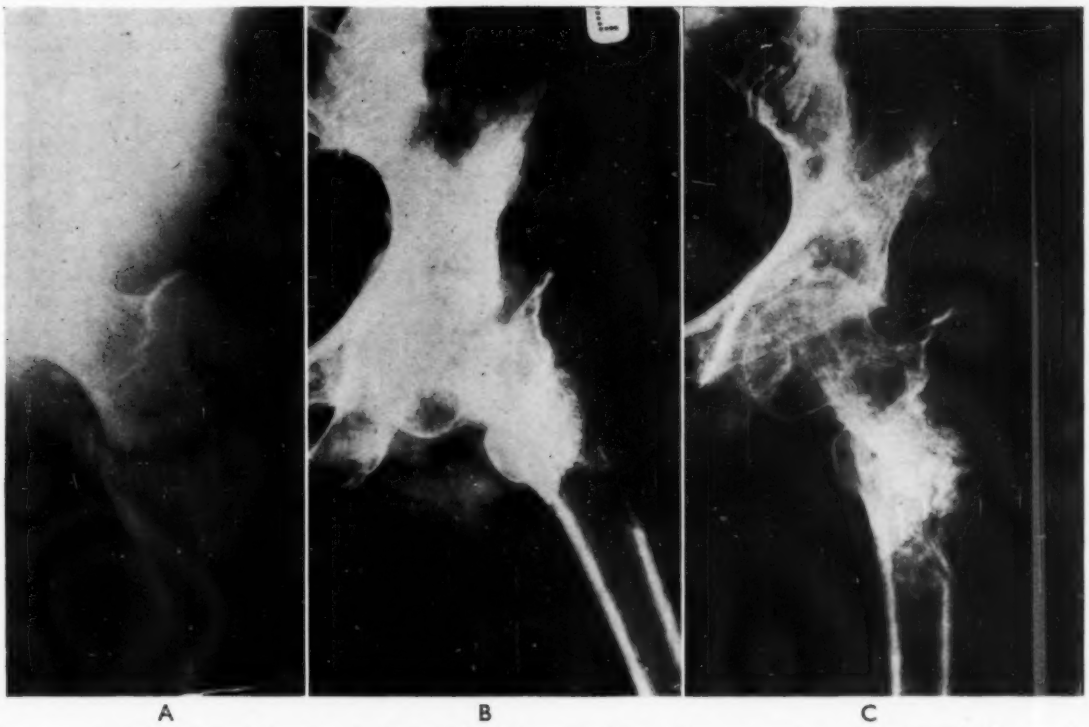
FIG. 250.—A, Apparent incomplete fracture of the great trochanter. B, Radiograph six weeks later showing complete fracture.

Type of Fracture.—We agree with Evans (1949) that the terms 'basal intertrochanteric' and 'pertrochanteric' are not clear and have no practical value. The all-embracing term 'trochanteric fracture' is preferred and of these there are two main types according to the direction of the fracture line.

1. The fracture line runs approximately parallel to the intertrochanteric line; coxa vara deformity may or may not be present, depending on the degree of trauma and comminution. Seventy-four cases (90 per cent). (*Fig.* 250.)

2. The fracture line runs approximately at right angles to the intertrochanteric line. Eight cases (10 per cent). (*Fig.* 251.)

It is also important to determine whether the fracture is comminuted. Comminuted fractures are unstable, reduction is often difficult, and most of the complications are confined to this group. Nine of the fractures are comminuted (11 per cent). (*Fig.* 252.)



A B C

FIG. 251.—A, Subtrochanteric fracture. B, Radiograph eight months later—union not present. C, Radiograph four months after insertion of iliac chips into the gap between the fracture surface, showing bony union.

Incomplete Fractures of the Great Trochanter. These fractures should be regarded with suspicion. In two patients a diagnosis of incomplete fracture of the great trochanter was made. Some weeks later, following a period of weight-bearing, persistent pain and limp led to a further X-ray examination which showed a complete trochanteric fracture (*Fig.* 250).

Results of Treatment.—*Table I* summarizes the methods of treatment and number of deaths in

FIG. 252.—A comminuted fracture treated conservatively. (The lateral radiograph, which was taken four months after the accident, has been retouched since the bony outlines are obscured by callus).

the two periods 1946–7 and 1948–9. It is suggested that the lower mortality in the second period is due to an increase in the use of internal fixation. A more detailed comparison is made of the two methods of treatment in *Table II*.

Table I.—TREATMENT OF TROCHANTERIC FRACTURES OF THE FEMUR

Treatment	1946–7		1948–9	
	No. of Fractures	Deaths	No. of Fractures	Deaths
Conservative	29	11	12	3
Internal Fixation	5	—	36	7
Total	34	11 (32 per cent)	48	10 (21 per cent)

It will be noted that internal fixation resulted in a shorter stay in hospital, a lower mortality, and fewer complications. Coxa vara is the commonest complication encountered; the lower incidence with internal fixation might be expected to lead to better functional results. It has not been possible to follow-up the conservatively treated cases, but of those treated by internal fixation 25 out of 34 patients obtained a good result (the patient walks well without sticks, the hip is free from pain, with more than two-thirds range of movement compared to the opposite hip and less than ½ in. of shortening of the limb). Two patients were unable to walk without considerable pain and needed sticks; there was less than two-thirds movement at the hip and more than ¾ in. of shortening. Four patients have been confined to bed since operation because of the following conditions: hemiplegia of long standing (two patients), senile psychosis (two patients). Three patients are untraced.

Table II.—TREATMENT OF TROCHANTERIC FRACTURES OF THE FEMUR

	Internal Fixation	Conservative
Number of fractures	41	41 Thomas splint 18 Fisk traction 11 Plaster spica 9 Well-leg traction 2 Bed rest 1
Average Age	69 years (range 29–90 years)	70 years (range 21–93 years)
Average duration of stay in hospital	6 weeks (12 days to 26 weeks)	14 weeks (2 weeks to 52 weeks)
Deaths	7 (17 per cent)	14 (34 per cent)
Cause of death	Bronchopneumonia 5 Hypostatic pneumonia 1 Strangulated femoral hernia 1 Infected bed-sore contributed to one death	Bronchopneumonia 6 Hypostatic pneumonia 1 Lobar pneumonia 1 Bed-sore and toxæmia 4 Pulmonary embolus 1 Cerebral thrombosis 1
Complications	5	14
Coxa vara	4	10
Refracture	–	2
Delayed union	–	1
Gangrene of leg	1 (amputation above knee)	1 (amputation above knee)

During the period under review there has been n increase in the use of internal fixation, so that during the year 1949 the great majority of trochanteric fractures were treated in this manner. Old age is no contra-indication to operation; in fact we believe that the increased mobility of the patient resulting from internal fixation is likely to reduce the incidence of those companions of immobility, respiratory infections, bed-sores, cerebral thrombosis, and pulmonary embolus.

Although we cannot present figures showing the relative frequency of senile psychosis, our impression is that this complication was less common in those treated by internal fixation.

In view of the rarity of non-union in association with trochanteric fractures, a short account of the one case in the series is presented. The patient was a woman aged 57 who sustained a low type of fracture. Well-leg traction was applied and at the end of three months, since union had not occurred, the limb was immobilized in a plaster spica. Nine months after the accident union was still not present, a marked gap being present between the fracture surfaces. The fracture was exposed through a lateral incision, the bone surfaces freshened and iliac chips packed into the gap, no attempt being made to correct the deformity. A plaster spica was reapplied. Union was present four months later (*see Fig.* 251).

Gangrene of the leg is a complication which so far as we are aware has not been noted before in association with trochanteric fractures. Our first example was a woman aged 75 who sustained a non-comminuted trochanteric fracture. A Fisk type of traction was applied. Two months later dry gangrene commenced in the big toe; the popliteal, anterior, and posterior tibial pulses were absent. An above-knee amputation was done a few days later. The femoral vessels showed extensive athermatous and arteriosclerotic changes; the lower part of the femoral artery was blocked by thrombus.

The second case was a woman aged 61 who developed gangrene of the foot in the fractured limb three days after the insertion of a nail-plate. Examination of the amputated leg suggested an embolic origin, the distal part of the popliteal artery being blocked by thrombus which extended down the anterior tibial artery. The arterial walls appeared healthy.

Recently we have met with two further cases of gangrene of the foot of the affected limb a week after the accident, and the other three months after. Both were examples of arteriosclerotic gangrene. It is suggested that the original trauma which causes the fracture and, to a lesser extent, manipulation of the limb may initiate thrombosis in an already markedly pathological vessel.

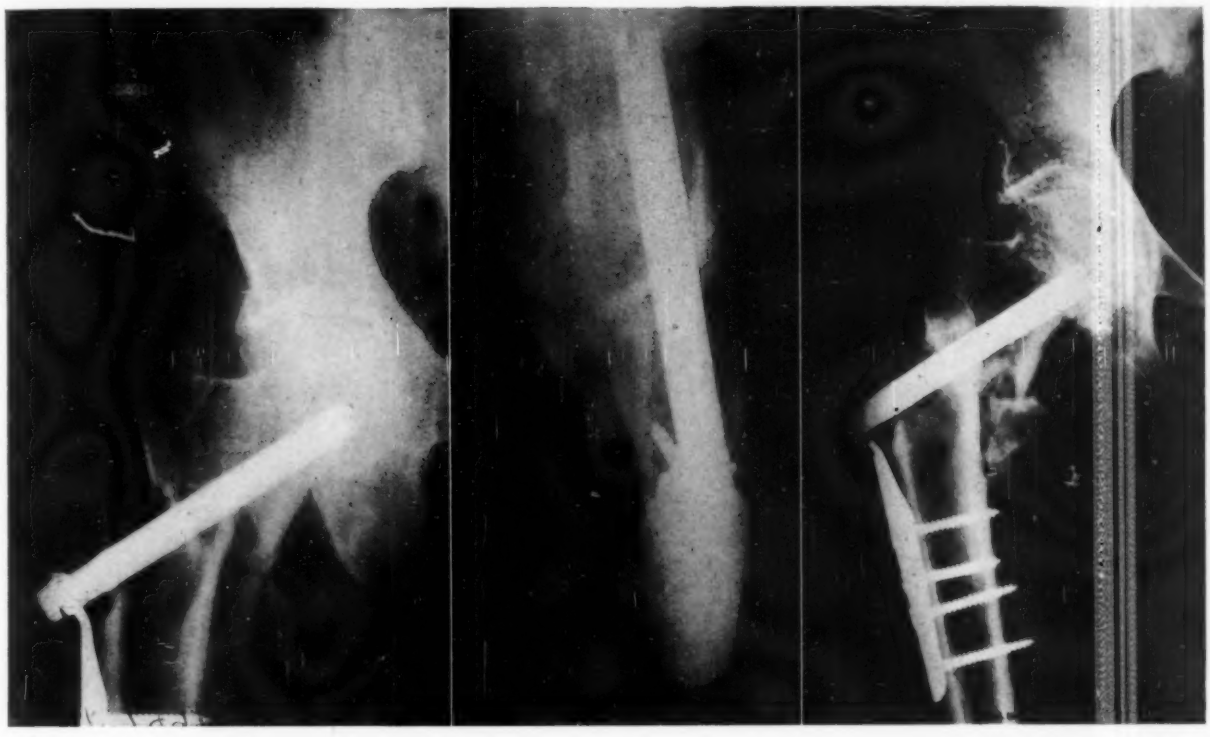
Operative Treatment.—Operation was performed by junior and senior members of the orthopædic staff (six surgeons). Pentothal, gas, and oxygen anæsthesia has been used. Blood-loss is usually small, and with careful hæmostasis blood transfusion should not be necessary.

In the majority of fractures, reduction has been obtained by traction on an orthopædic table combined with moderate abduction and internal rotation. In a number of comminuted fractures, a neutral position or some degree of external rotation has been necessary.

The McKee (1944) nail-plate has been used in all cases. This consists essentially of a cannulated

Smith-Petersen nail with a detachable plate fixed to the head of the nail by a self-locking nut. The Smith-Petersen nail is inserted over a previously positioned guide wire using the same technique as with fractures of the femoral neck. The angled attachment of the plate is bent to the required angle, attached to the pin by a nut and fixed to the femoral shaft by four screws.

Complications of Internal Fixation.—1. *Sepsis.*—One patient had a small discharging sinus for two months following operation. One patient complained of local pain eighteen months after operation. A loose screw showed in the radiograph. At operation when the nail-plate was removed, a small collection of pus was found round the loose screw. Primary healing occurred.



A B C

FIG. 253.—A, B, Comminuted fracture immobilized by nail-plate with imperfect reduction. Cortical overlap and slight internal rotation deformity was present in the lateral radiograph. C, Five months later a moderate coxa vara deformity was present with a fracture of the plate at the angle; bony union was sound. The plate fracture occurred between four and five months after the accident.

The operative technique differs from that used in nailing fractures of the femoral neck in three ways:—

1. In order to prevent splintering of the brittle lateral femoral cortex, a hole, 1 cm. in diameter, is drilled at the site where the nail is to be inserted.

2. The point of entry of the nail should be at the level of, or a little below, the lesser trochanter, i.e., well below the fractured and possibly comminuted great trochanter.

3. The distal part of the nail should lie not less than ½ in. from the articular surface of the femoral head. In comminuted fractures this is important, since there is often some collapse of the fracture site which results in shortening of the neck and penetration of the nail into the joint.

One month after operation, patients are instructed to walk with the aid of crutches; where the fracture has never been displaced, walking with crutches is allowed earlier, whereas in comminuted fractures walking is delayed six to eight weeks. Crutches are discarded after three or four months when radiological union is present, and weight-bearing with the aid of two sticks is continued for another four weeks.

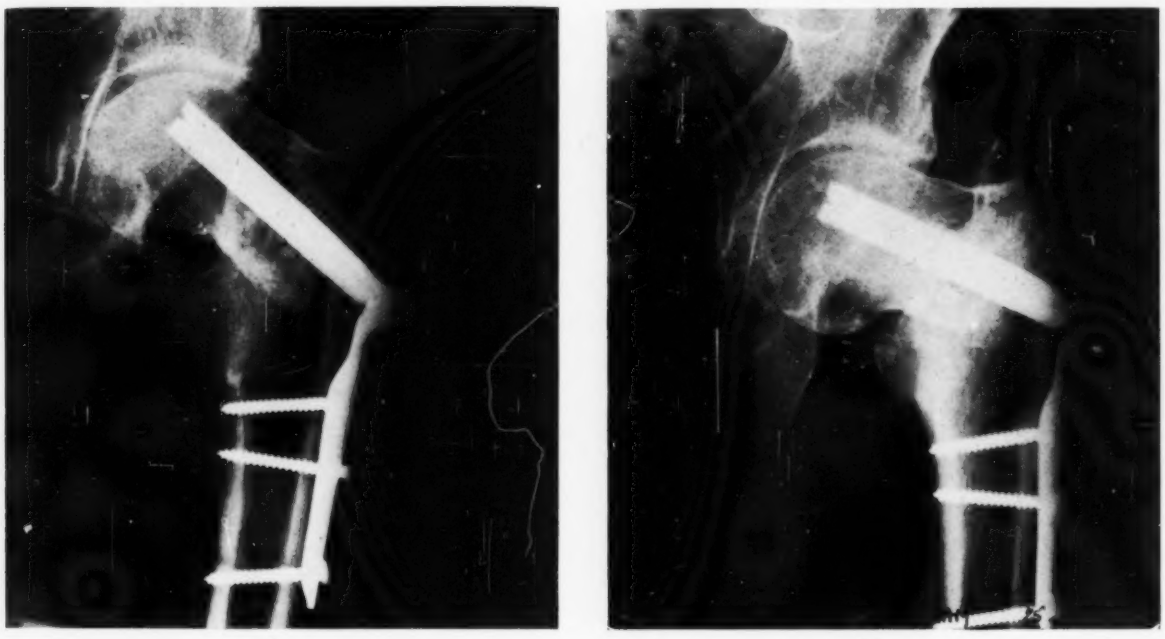
2. *Coxa Vara.*—Four patients. This condition was associated with the following changes in the nail-plate: (*a*) Fracture of the plate at the angle —*Case* 1 (*Fig.* 253), *Case* 2 (*Fig.* 254). (*b*) Bending of the nail—*Case* 3 (*Fig.* 255). (*c*) The nail cut out through the posterosuperior surface of the neck —*Case* 4 (*Fig.* 256).

All these four fractures were comminuted in varying degrees. In *Cases* 2 and 3 varying degrees of cortical overlap were present as seen in the lateral radiograph and in *Cases* 1 and 3 there was an internal rotation deformity of the distal part of the femur suggesting that reduction would have been better with the limb in external rotation. The complication in *Case* 4 was probably due to a short nail cutting out through the anterosuperior surface of the femoral neck and thus causing an external rotation deformity of the limb.

3. *Penetration of the Nail into the Joint* (*Fig.* 257). —This occurred one month after operation, when the patient began to walk with crutches. A lateral radiograph at the time of operation showed cortical overlap and well-marked internal rotation deformity. The nail was placed less than 1 cm. from the articular margin.

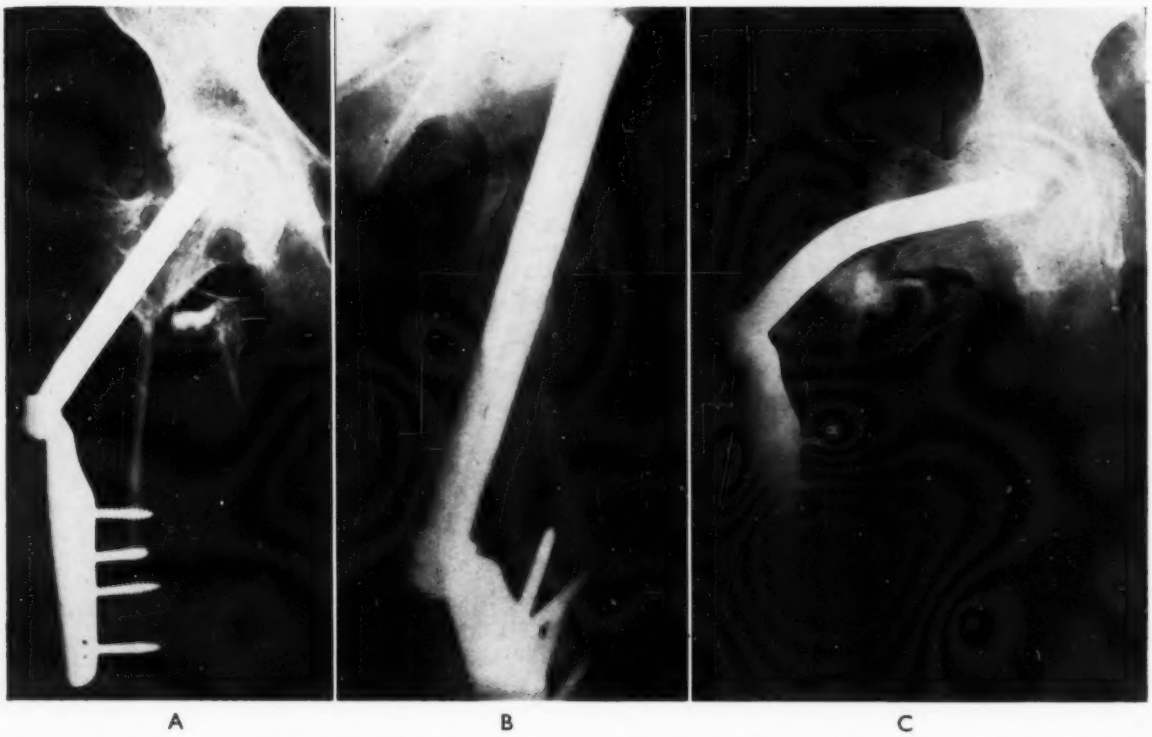
In 4 patients, internal fixation was abandoned owing to failure of reduction. All these fractures were comminuted. Two could not be reduced, though various positions of the limb were tried. In the remaining 2 cases a circumferential wire was applied around the fragments and the limb immobilized on a Thomas splint.

An opportunity to examine the femur at the post-mortem occurred in one patient who died six weeks after operation as a result of a respiratory



A B

FIG. 254.—A, Radiograph immediately after fixation. Cortical overlap was present in the lateral radiograph. B, Radiograph five months later, showing coxa vara and fracture of plate.



A B C

FIG. 255.—A markedly comminuted fracture. Nailing and reduction was apparently good in the anteroposterior radiograph A, but marked cortical overlap and internal rotation deformity showed in the lateral radiograph B. C, Radiograph five months later showed bending of the nail. This patient was discharged from hospital six weeks after operation. As the result of imperfect home supervision and a mild senile dementia, she began to bear weight six weeks after operation, contrary to instructions.

infection. The fracture was of the non-comminuted type 1. Reduction was good, the nail-plate was in good position, and the fracture felt firm after the removal of the metal. The nail track was seen to be

FIG. 256.—A, Reduction of the fracture and direction of the nail were satisfactory. (Unfortunately only the lateral view is suitable for reproduction.) B, C, Radiograph one month later. The nail has cut out through the anterosuperior surface of the femoral neck. This was probably due to a short nail which obtained an imperfect hold of the proximal fracture segment.

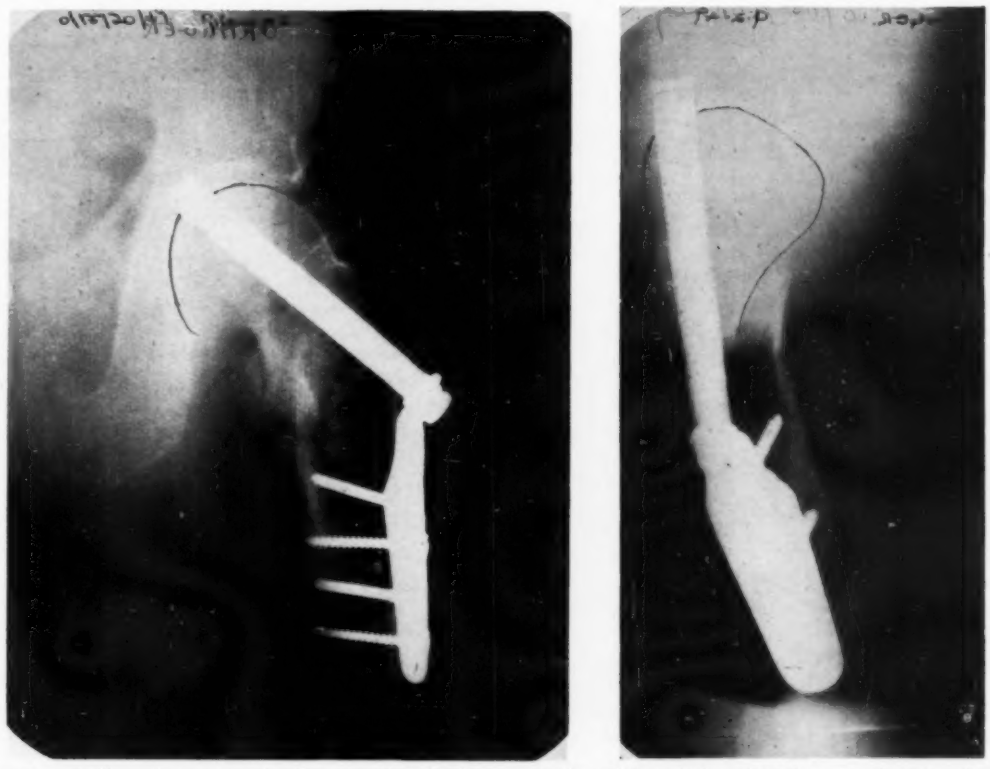


FIG. 257.—Radiographs one month after operation showing penetration of the nail into the joint. The lateral radiograph shows marked cortical overlap and internal rotation deformity.

lined by a smooth, shining, fibrous surface, which on section proved to be a condensation of bony vascular fibrous tissue. Endosteal callus was present at the fracture site across which new bone trabeculæ had already formed. No chondroid metaplasia was seen in the callus, a fact attributable to the perfect immobilization secured by the nailing.

CONCLUSIONS

1. Trochanteric fractures are associated with a high mortality whatever treatment is adopted.

2. The introduction of internal fixation has resulted in a lowered mortality, a better anatomical result, and a shorter stay in hospital.

3. Comminution of the fracture is frequently associated with complications.

a. Some severely comminuted fractures may be mechanically unsuitable for internal fixation (*see Fig.* 252). A lateral radiograph is necessary before a decision can be made on the appropriate method of treatment.

b. These fractures are often difficult to reduce, and some external rotation of the limb may be necessary to obtain reduction. If internal fixation is used and the fracture is imperfectly reduced, with cortical overlap and rotational deformity, trouble should be expected. Coxa vara will develop or the neck will shorten as the result of telescoping of the upper fragment into the lower.

c. In spite of comminution, union in good position can usually be obtained and it has not been the practice to fix these fractures in a position of coxa vara as recommended by Mervyn Evans (1949). Union will take considerably longer than the normal ten to twelve weeks, and weight-bearing should be delayed until indisputable evidence of radiological union is present.

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EXPERIMENTAL SURGERY

THE INFLUENCE OF CORTISONE ON THE REPAIR OF EXPERIMENTAL FRACTURES IN THE RABBIT

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THE clinical use of cortisone in a variety of conditions has directed attention to the profound effects of this substance on cell multiplication and tissue repair. Not only does the cellular infiltration of the synovial membrane in rheumatoid arthritis regress under treatment (Hench, Kendall, Slocumb, and Polley, 1949), but it is frequently found that skin incisions in cortisone-treated patients fail to repair normally (Plotz, Howes, Meyer, Blunt, Lattes, and Ragan, 1950). Moreover, patients suffering from Cushing's syndrome, with an intrinsic oversecretion of this type of adrenocortical hormone, show a general inhibition of cellular reparative processes as shown by occasional failure of wound healing (Loeb, 1950), atrophic cutaneous striæ, and particularly by osteoporosis due to suppression of normal osteoblastic activity (Sissons, 1951).

A number of experimental investigations of the effects of cortisone on tissue repair have been carried out. The findings of one group of workers (Howes, Plotz, Blunt, and Ragan, 1950), are representative, and show a retardation of repair both in skin wounds and experimental fractures in rabbits. In a paper dealing specifically with the effect on fracture repair (Blunt, Plotz, Lattes, Howes, Meyer, and Ragan, 1950), these investigators described an inhibition of the development of fracture callus, but their observations extended only over a short period of time and their histological findings were not presented in detail.

In the present investigation, closed fractures of tibia were used as a convenient subject for the study of tissue repair. This choice was made because the closed wound was not subject to bacterial contamination from outside, the progress of repair could be followed by means of radiographs as well as by histological study, and because a healing fracture affords a specially favourable medium for demonstration of the maturation and differentiation of mesenchymal cells.

PLAN OF THE EXPERIMENTS

Two groups of young rabbits were used as experimental animals. In one the daily dose of cortisone administered was 20 mg./kg.; in the second it was 10 mg./kg. These doses are somewhat less than that used by Blunt et al., but, on a basis of weight, considerably larger than the usual human therapeutic dose. Each cortisone animal had a control of similar weight which was maintained under identical conditions. Fracture of the tibia was performed under anæsthetic at the junction of the upper and middle

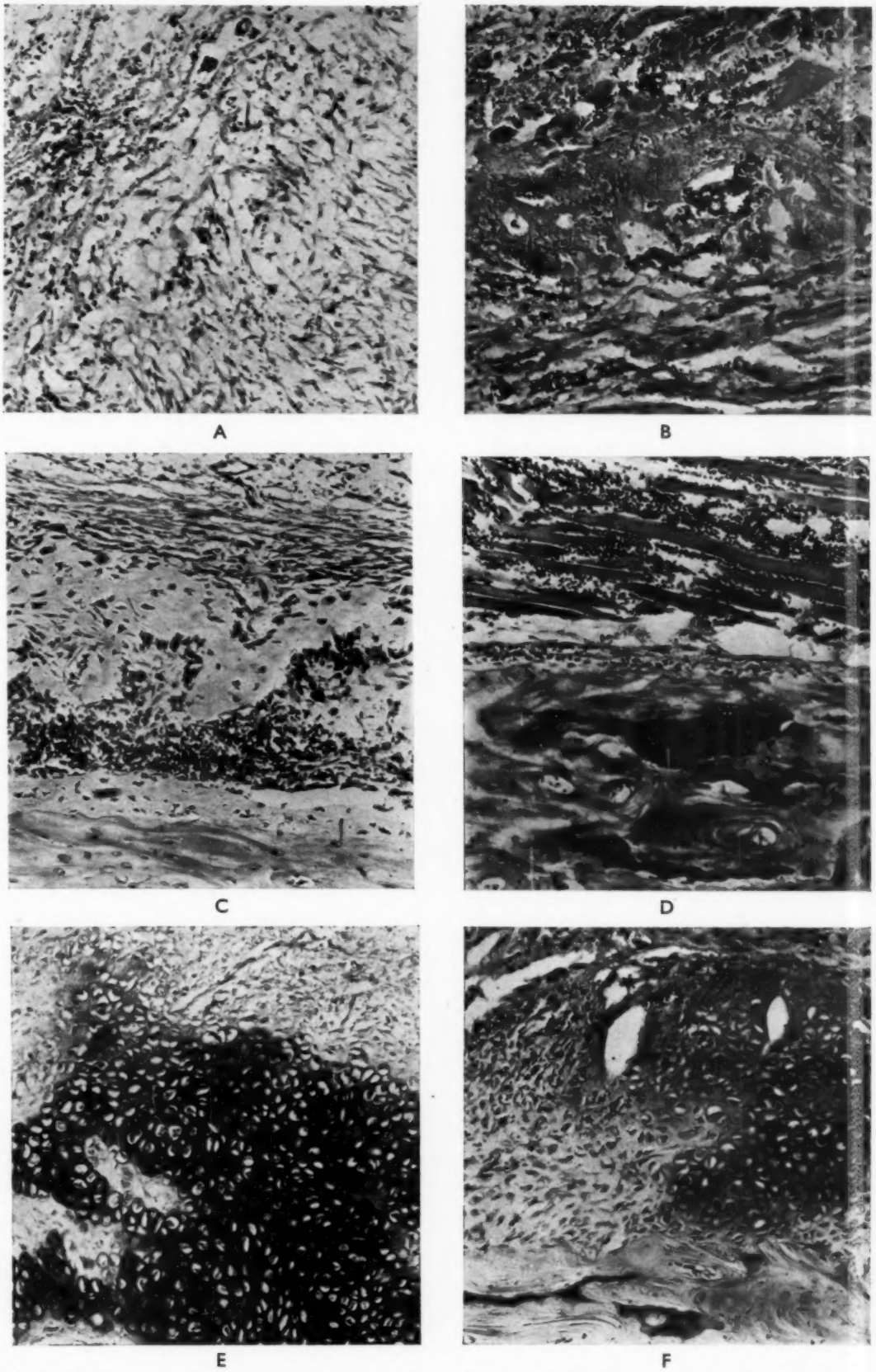


FIG. 258.—HISTOLOGICAL APPEARANCES OF FRACTURE CALLUS IN CONTROL AND CORTISONE-TREATED ANIMALS AT 4–5 DAYS. A, Control. Development of granulation tissue. (× 105.) B, Cortisone (20 mg.). Blood-clot but no granulation tissue. (× 105.) C, Control. Active formation of periosteal bone. (× 105.) D, Cortisone (20 mg.). Comparable area of periosteum without cellular proliferation or bone formation. (× 105.) E, Control. Conspicuous cartilaginous callus. (× 95.) F, Cortisone (10 mg.). Minimal development of cartilaginous callus. (× 105.)

thirds, and animals were sacrificed at convenient intervals for histological study. In the 20-mg. group the experimental animals showed marked loss of weight and few appeared likely to survive longer than twelve days after fracture. In spite of weight loss the experimental animals in the 10-mg. group did not show this mortality and survived up to the time of complete union of the fractures in their controls.

In all animals the mobility of the fractured bone was determined each day, and when animals were killed a detailed post-mortem examination was carried out. In each animal, processes of repair were studied by preparing slab-radiographs of the involved bones according to the technique already described by one of the present authors (Sissons, 1950), and then preparing histological sections from the same tissues.

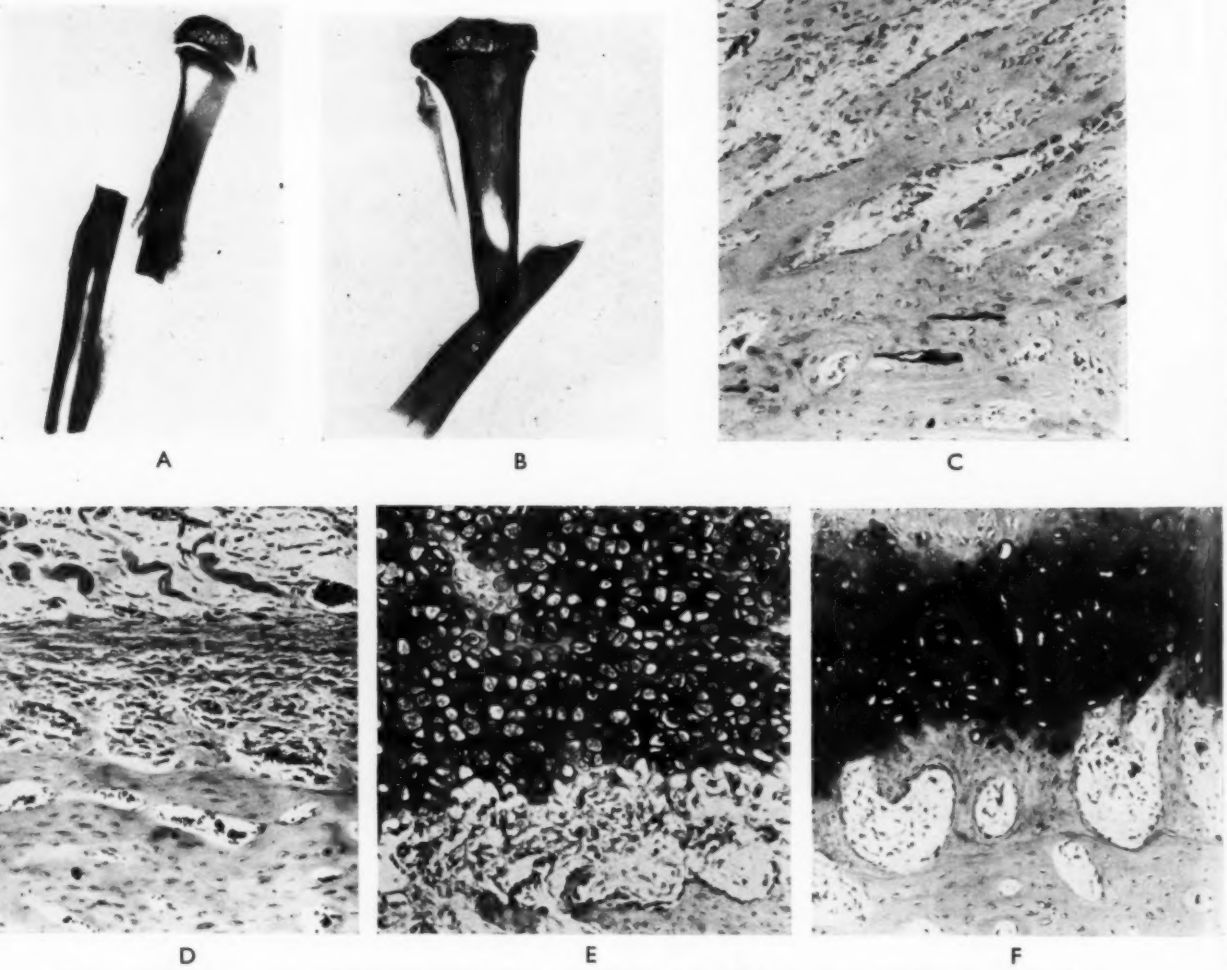


FIG. 259.—RADIOGRAPHIC AND HISTOLOGICAL APPEARANCES OF CONTROL AND CORTISONE-TREATED ANIMALS AT 7–8 DAYS.
A, Control. Calcification of callus in subperiosteal region. (× 1·2.) B, Cortisone (20 mg.). Absence of calcification at fracture site. (× 1·2.) C, Control. Thick deposit of periosteal bone. (× 90.) D, Cortisone (20 mg.). Comparable area of periosteum with minimal bone formation. (× 90.) E, Control. 'Hypertrophic' cartilaginous callus undergoing vascularization. (× 90.) F, Cortisone (10 mg.). Inert cartilaginous callus showing bony transformation. (× 90.)

RESULTS

By four days, the fracture in the normal animals became firm to palpation, and in ten days the bones showed clinical union. In contrast, the cortisone-treated animals retained a completely mobile fracture throughout the entire period of study, or at most showed a slight resistance to movement towards the end of the experiment.

At post-mortem the cortisone-treated animals showed a marked diminution in adrenal and splenic weight. As well, there was a marked degree of general muscle atrophy. During life it was noted that several of the 20-mg. group developed a severe diarrhœa. Arrest of epiphysial bone growth was noted to occur in all the experimental animals, and during the course of the experiment the bones of the cortisone-treated animals showed some generalized rarefaction in comparison with their stouter and denser controls. Details of epiphysial changes and of muscle repair, which were studied in detail, are being presented elsewhere.

After about four days the control animals showed progressive calcification of fracture callus in radiographs. There was complete bridging of the fracture by calcified callus at about twelve days, and at about eighteen to twenty days, bony union of the fracture was complete.

In the cortisone-treated animals the fracture site showed no calcification at all until about eleven days, and even after three weeks there was merely a very slight degree of periosteal reaction in the neighbourhood of the fracture.

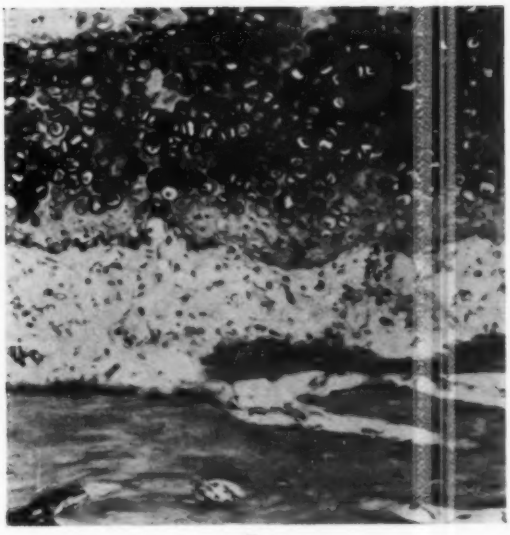
The following is an outline of the histological processes of fracture repair in the normal animals; it is essentially the same as the findings of Ham (1930) in an experimental study of normal bone repair.

Widespread hæmorrhage takes place at the time of fracture, and the immediate response to it is the invasion of the hæmatoma by developing vascular granulation tissue. This is a conspicuous histological finding in the early days after fracture, and involves surrounding muscle in addition to the endosteal and periosteal tissues. At about four to five days the deposition of periosteal bone in the neighbourhood of the fracture becomes apparent, and the proliferating periosteal tissue and nearby granulation tissue show the development of areas of cartilage. This periosteal bone and cartilaginous callus increases in extent and joins the bone ends together. At about seven days the cells of the proliferating cartilage begin to show a 'hypertrophic' appearance, and this is followed by calcification, vascularization, and progressive bony replacement of this tissue. These processes are active at ten days, and by about sixteen days the bridging callus consists entirely of bony trabeculæ. Further changes take the form of remodelling and consolidation of this bony structure.

A

B

C

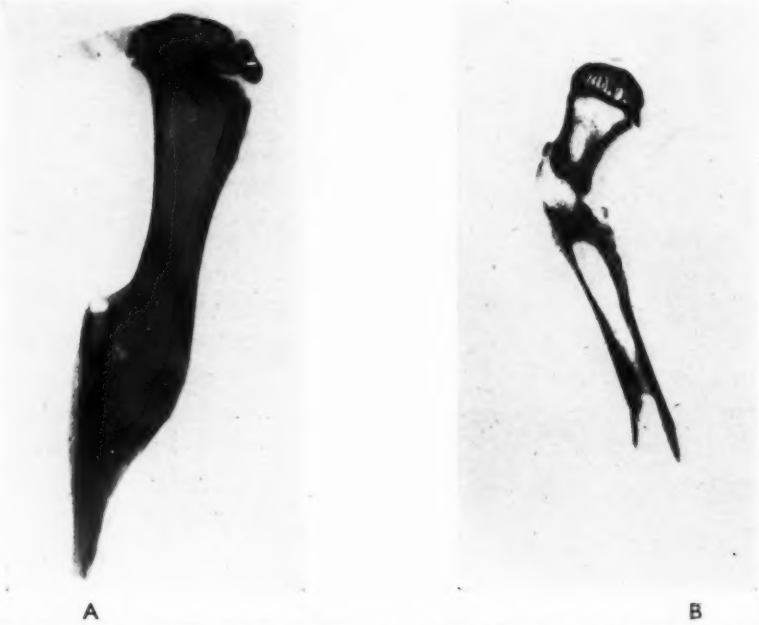


D

FIG. 260.—RADIOGRAPHIC AND HISTOLOGICAL APPEARANCES OF CONTROL AND CORTISONE-TREATED ANIMALS AT 11–12 DAYS. A, Control. Bridging of fracture gap by calcified callus. (× 1·2.) B, Cortisone (10 mg.). Slight periosteal reaction only. (× 1·2.) C, Control. Cartilaginous callus now replaced by bone trabeculæ. (× 105.) D, Cortisone (20 mg.). Inert cartilaginous callus without vascularization or bony replacement. (× 105.)

In the cortisone-treated animals all these processes are delayed. At four days, for instance (*see Fig.* 258), there is little development of granulation tissue, and almost no periosteal bone or cartilaginous callus. Indeed, with a dose of 20 mg./kg., these features of the repair process are completely suppressed at this stage. At seven to eight days (*Fig.* 259) a minimal degree of periosteal reaction may be present, and some cartilaginous callus has developed, while at twelve days (*Fig.* 260), when in normal animals a bulky mass of cartilaginous callus is largely replaced by bone, the cortisone-treated animals show only small areas of cartilaginous callus. Moreover, the cartilage differs from that of normal callus in that it fails to show 'hypertrophic' cells, and is not vascularized and replaced by bone. At this stage, and at later periods (*Figs.* 261, 262), the cartilage shows degenerated areas where the normal intense metachromatic staining of the cartilage matrix is lost, and where the tissue undergoes marked fragmentation. The cartilage which fails to undergo normal maturation and vascularization appears to undergo a process of gradual conversion to bone (*Fig.* 262, C), which is not seen in the control material. In the course of this process the cartilage matrix gradually loses its metachromatic staining reaction, and the cartilage cells become converted into osteocytes. In some areas a narrow zone of this changed tissue is seen to surround the bulk of the cartilaginous callus (*Fig.* 260, D).

Details of the findings in each experimental animal are set out in *Table I.*



A B

FIG. 261.—RADIOGRAPHIC APPEARANCES OF CONTROL AND CORTISONE-TREATED ANIMALS AT 19–21 DAYS.
A, Control. Complete bony union. (× 1·2.) B, Cortisone (10 mg.). Minimal periosteal reaction at site of fracture. (× 1·2.

DISCUSSION

These results confirm the inhibition of repair processes by cortisone that has been noted by other authors, and give for the first time a detailed presentation of the histological findings for bone tissue.

Not only is the development of granulation tissue inhibited but the formation of specific skeletal structures is retarded. When cartilaginous callus is ultimately produced, its maturation and bony replacement do not take place in the normal fashion.

It is of interest to note that the process of bony transformation of cartilaginous callus in the absence of its vascularization, which occurs in these cortisone-treated animals, but which is not normally seen in bone repair in mammals, is a characteristic feature of the process in lower vertebrate forms, as described by Pritchard and Ruzicka (1950). These two authors, and also Ham (1930), have expressed the view that the differentiation of the proliferating tissue of fracture callus in the direction either of bone or cartilage may be controlled by the degree of vascularity of the tissue, a high degree of vascularity favouring the development of bone, and a relative avascularity favouring that of cartilage. The effect of cortisone on the inhibition of vascular development generally, and its particularly effective inhibition of both the development of bone trabeculæ and the vascularization of cartilaginous callus are thus perhaps immediately related, although the present writers do not feel that the postulated influence of vascularity on the differentiation of fracture callus has yet been firmly established.

It should be pointed out that in common with other effects of cortisone, the inhibition of fracture repair cannot be produced with equal facility in all species of animals. In an experiment with rats, using the same dosage of cortisone per unit of weight, the present authors found far less interference with fracture repair than is described in the present paper. It is not known on what this marked species variation depends, although because of cortisone's action in depleting adrenal ascorbic acid it has been suggested that the production of tissue changes by cortisone

night be related to the particular species' sensitivity to vitamin C depletion. What is known of the human responses to therapeutically administered cortisone, and of the impaired repair processes in adrenal hormone. This may well be the metabolic basis of the interference with the proliferation and differentiation of mesenchymal cells described in the present paper.

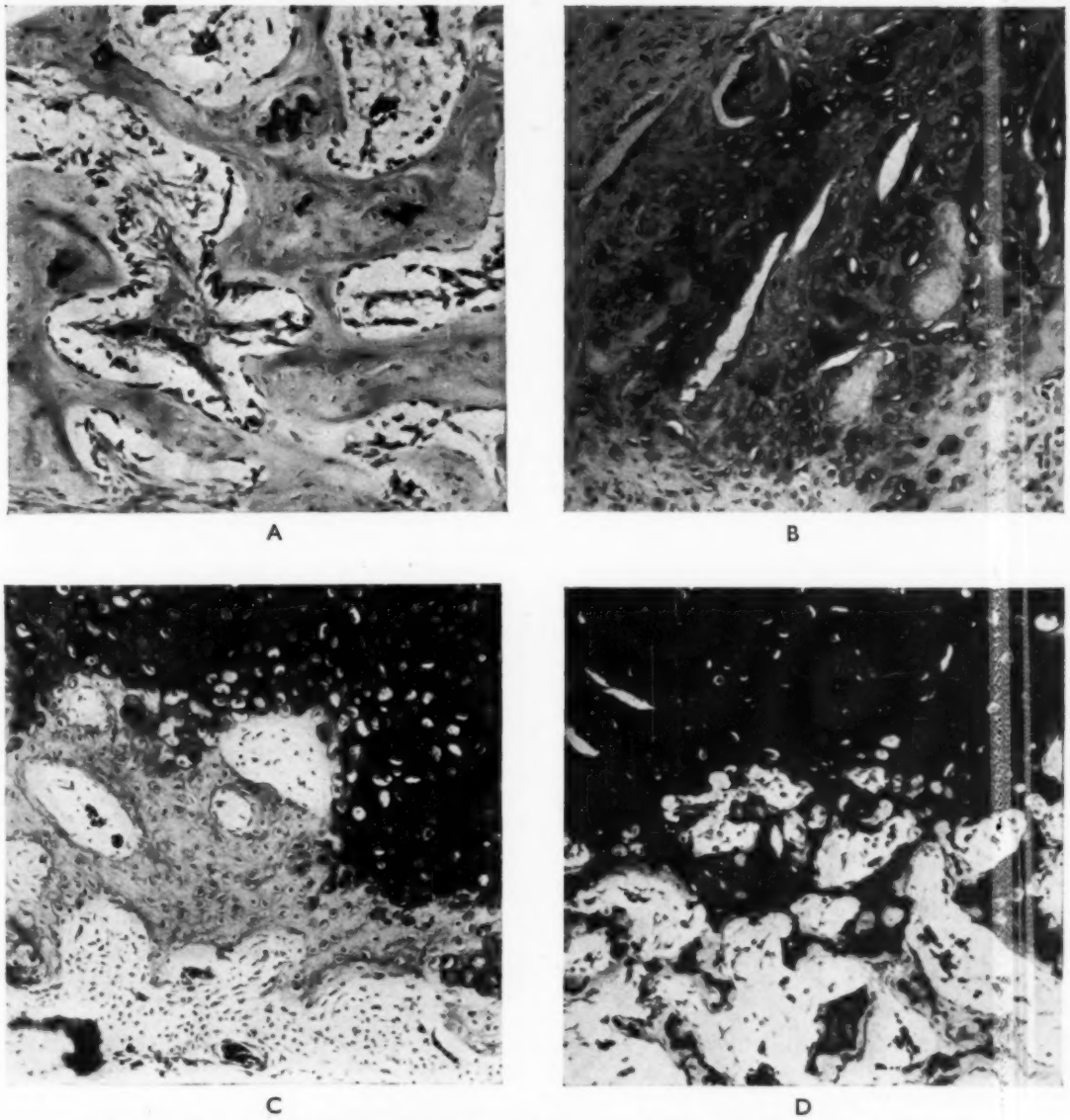


FIG. 262.—HISTOLOGICAL APPEARANCES OF CONTROL AND CORTISONE-TREATED ANIMALS AT 19–21 DAYS.
A, Control. Thickened bone trabeculæ in consolidating callus. (× 105.) B, Cortisone (10 mg.). Degenerated cartilaginous callus showing fragmentation and loss of metachromatic staining. (× 110.) C, Cortisone (10 mg.). Cartilaginous callus showing direct conversion to bone in the absence of vascularization. (× 96.) D, Cortisone (10 mg.). Commencing vascularization and bony replacement of cartilaginous callus. (× 110.)

patients suffering from Cushing's syndrome, suggests that man possesses at least some of the features of the rabbit's response as described above.

The following features are common—under these circumstances—in the two species: Inhibition of development of vascular granulation tissue, osteoporosis with impaired ability for bone repair, cessation of growth, and muscular weakness and atrophy. It is the view of Albright (1942–3) and Reifenstein (1950) that such diverse manifestations can be explained by a failure of protein synthesis—the 'anti-anabolic' effect of the cortisone type of

SUMMARY

An experimental investigation of the effects of cortisone on the repair of experimental fractures in the rabbit is described.

At a daily dose level of 10 mg. or 20 mg. per kg. it is found that fractures fail to unite after as long as twenty-one days, and that all the histological processes of repair in bone are retarded.

In particular, attention is directed to the absence of periosteal bone formation, and to the abnormal histological processes taking place in cartilaginous callus.

Attention is directed towards the similarity between these features of experimental cortisone administration in the rabbit, and the inhibition of

Table I.—Details of Findings in Experimental Animals

Rabbit No.	No. of Days after Fracture	Cortisone Dose	X-ray Appearances (Calcification)	Histology			Remarks
				Granulation Tissue	Periosteal Bone	Cartilaginous Callus	
9	3	20 mg.	None	—	—	—	
10	4	Control	None	++	++	++	
13	4	20 mg.	None	+	—	—	
14	4	Control	None	+++	++	+	
7	4	20 mg.	None	—	—	—	
18	4	10 mg.	None	+	+	+	
17	4	Control	None	+++	++	++	
8	5	Control	Slight	+++	++	++	
2	7	20 mg.	None	+	+	+	Atypical cartilaginous callus
5	7	Control	Slight	++	+++	+++	Beginning replacement of cartilaginous callus by bone
11	7	20 mg.	None	+	+	+	Atypical cartilaginous callus, but no bony replacement
12	7	Control	Slight	++	+++	+++	Beginning replacement of cartilaginous callus by bone
26	8	10 mg.	Slight	+	+	+	Atypical cartilaginous callus, but no bony replacement
19	8	Control	Considerable	++	+++	+++	Active replacement of cartilaginous callus by bone
29	11	10 mg.	Slight	+	+	++	Atypical cartilaginous callus with beginning bony replacement and some direct conversion to bone
24	11	Control	Considerable	++	+++	+++	Active replacement of cartilaginous callus
1	12	10 mg.	Slight	+	+	++	Atypical cartilaginous callus, but no bony replacement. Some direct conversion to bone
4	12	Control	Considerable	++	+++	+++	Active replacement of cartilaginous callus by bone
23	14	10 mg.	Slight	++	+	++	Atypical cartilaginous callus with slight bony replacement and some direct conversion to bone
25	14	Control	Almost complete	++	+++	+++	Active replacement of cartilaginous callus by bone
21	17	10 mg.	Slight	—	—	+	No bony replacement of atypical cartilaginous callus. Some direct conversion to bone
22	17	Control	Almost complete	+	+++	—	Cartilaginous callus almost completely replaced by bone
28	18	10 mg.	Moderate	+	—	++	Slight bony replacement of atypical cartilaginous callus. Some direct conversion to bone
20	19	Control	Almost complete	—	+++	—	Cartilaginous callus completely replaced by bone. Consolidation of bony callus
15	21	10 mg.	Slight	—	+	++	Slight bony replacement of atypical cartilaginous callus. Some direct conversion to bone
16	21	Control	Complete	—	+++	—	Cartilaginous callus completely replaced by bone. Consolidation of bony callus.

tissue repair that is seen in patients either suffering from Cushing's syndrome or receiving therapeutic doses of cortisone.

Acknowledgement.—The cortisone used in this work was provided from a generous gift made jointly to the Medical Research Council and the Nuffield Foundation by Merck & Co. Inc.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF CONGENITAL SACROCOCCYGEAL TERATOMA

By KEITH D. ROBERTS, Birmingham

Congenital sacrococcygeal tumours are not common, and it has been thought worth while to record the following case.

CASE REPORT

A male infant was admitted to hospital on Nov. 6, 1949, seven hours old, as a case of spina bifida with meningocele. The baby was the youngest of six, all the others being normal. The birth weight was 10 lb. and the child was said to have passed meconium per rectum prior to admission.

Abdominal Examination.—No abnormality found.

Rectal Examination.—The anal canal and rectum were intact and patent; there was no apparent connexion between rectum and tumour; the tumour did not appear to extend into the sacral hollow, and meconium was present on the examining finger.

Radiological Examination.—The pelvis appeared normal; no bone changes were seen and the tumour appeared to consist entirely of soft tissues.

A diagnosis of sacrococcygeal teratoma was made. The skin overlying the tumour was so thin that early operation to avoid ulceration and infection was indicated

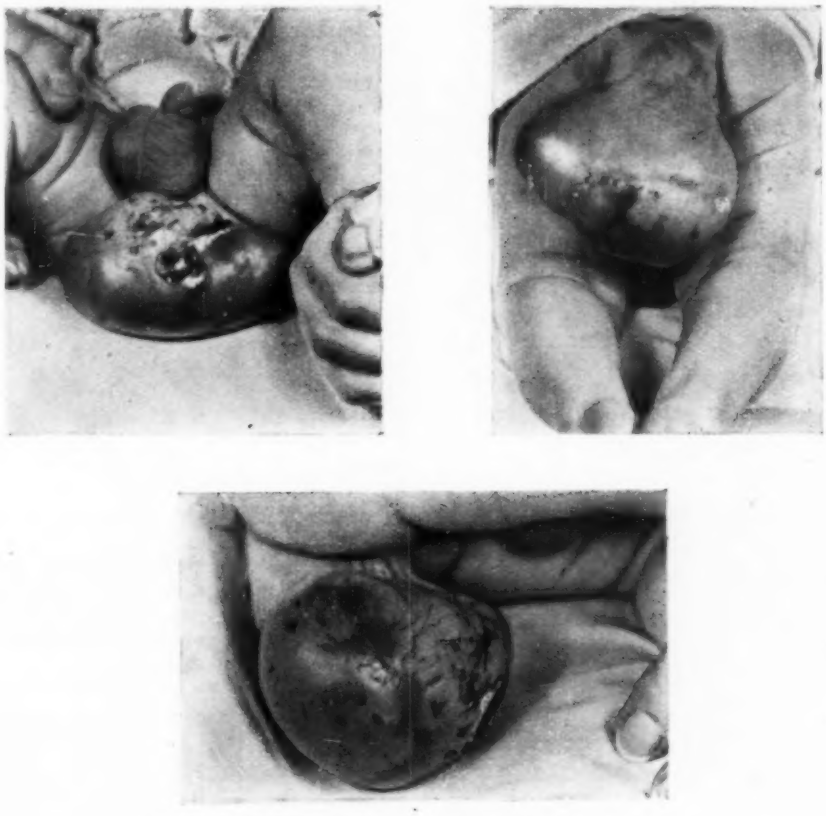

Fig. 263.—Photographs showing the situation of the tumour.

Clinical Examination.—The infant was well developed and cried lustily, moving all its limbs. A rounded swelling, the size of an orange, was attached by a pedicle 6 cm. long and 5 cm. in diameter, to a base roughly elliptical in shape, of diameter 8 cm. longitudinally and 5 cm. transversely, the base being situated over the sacrum, as shown in *Fig.* 263. There was no palpable deficiency in the neural arches. The tumour felt cystic, but did not empty with gentle pressure, and no fluctuation could be obtained between the tumour and the anterior fontanelle. Neither muscular twitchings nor peristalsis were seen in the tumour, from which it was deduced that there was no muscle or bowel within the the tumour mass. There was no palpable bony structure.

and was carried out on Nov. 14 after commencing a course of systemic penicillin.

Operation.—Performed under open ether and oxygen anæsthesia, with the baby lying prone. A vertical elliptical incision 10 cm. long was made surrounding the base of the pedicle. The lower borders of gluteus maximus were exposed on either side and the posterior wall of the rectum was found to present anteriorly in the wound, there being no attachment of the levatores ani to the coccyx. Numerous large vessels required ligation. The pedicle of the tumour was found to extend on to the anterior aspect of the coccyx and lower sacrum and terminated as a small cyst. The anterior dissection was not difficult and removal of the coccyx was not

performed. The posterior attachment of the tumour was found to extend down to the periosteum of the sacrum. The wound was insufflated with penicillin powder and completely closed, without drainage, with interrupted nylon sutures.

The post-operative progress was uneventful; the wound healed well and the baby was discharged one month after admission. When last seen on June 20, 1950, the scar was sound and there was no evidence of recurrence.

THE SPECIMEN.—On hemisection the tumour was found to consist of one large and numerous small cysts (*Fig.* 264) containing clear fluid. No formed elements were present.

FIG. 264.—Sagittal section of the tumour.

Microscopical report: The tumour is composed of a number of cysts, lined by a variety of epithelia often varying within the same cyst. The supporting stroma consists mainly of vascular connective tissue with several bundles of plain muscle, and collections of fœtal fat. This is a benign cystic sacrococcygeal teratoma.

COMMENT

Calbet, quoted by Hundling (1924), states that congenital tumours of the sacrococcygeal region occur once in every 34,582 births; teratomas represent only a very small percentage of these. Ewing (1928) reported that one-third of fœtuses with such tumours are born dead, and that 90 per cent of the rest die soon after birth. Chaffin (1939) found that 75 per cent of patients with sacrococcygeal teratomas were females. Owing to their size it is common for the tumours to cause dystocia.

Willis (1947) points out that the teratomas are essentially similar to retroperitoneal teratomas in general, and must be distinguished from caudally attached twins, supernumerary limbs, and residues of the post-anal gut and neural canal. He states that the teratomas never show any orderly arrangement of parts and never contain a vertebral axis. He attributes the origin of the tumours to release of islands of embryonic tissue from the action of the primary organizer.

The most important differentiation in diagnosis has to be made from meningocele. DeVeer and Browder (1937) make the following points in this connexion. A meningocele is smaller, covered with translucent membrane, not skin, and does not displace the rectum. There is usually some evidence of communication between a meningocele and the remainder of the subarachnoid space, such as expansion on crying or bulging of the fontanelle on compression of the tumour. Neurological signs in the lower extremity favour a diagnosis of meningocele.

Sacrococcygeal teratomas are liable to the following complications:—

1. *Malignancy*: 15 per cent of cases undergo malignant change. Usually only one tissue is involved, and the neoplasms are most often neural in origin or are papillary carcinomas. According to Oldham (1950), the more lowly organized tumours are more prone to malignant change, and the presence of bony structures, muscular twitchings, or intestinal loops within a teratoma favours its being benign.

2. *Ulceration and sloughing*: These may result in fistula formation.

3. *Intestinal obstruction and retention of urine*: These are liable to develop if the tumour expands upwards into the pelvis.

4. *General effects*: Very rarely hypersecretion of an endocrine tissue in the teratoma may produce some metabolic disturbance.

Riker and Potts (1948) believe that early complete excision of the tumour is the method of choice, especially in view of the danger of ulceration and infection. If histological examination reveals the presence of malignant elements, post-operative radiation should be employed. These authors quote Pearse (1921) in emphasizing the use of the posterior approach for accessibility, control of hæmorrhage, avoidance of the peritoneal cavity, and early detection of local recurrences in the scar.

I am indebted to Dr. L. W. Proger, of the Royal College of Surgeons, for the pathological report; and to Mr. C. South, of St. Chad's Hospital, Birmingham, for permission to treat this case. I also acknowledge the help given by Dr. Frances Braid with the medical care of this patient.

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SACCULAR ANEURYSMS OF THE INTERNAL CAROTID ARTERY IN THE CAVERNOUS SINUS OCCURRING BILATERALLY

By VALENTINE LOGUE, London

If one may judge from the reports in the literature, of which there are only six (Blane, 1800; Sosman and Vogt, 1926; Bozzoli, 1937; Dandy, 1937; Jefferson, 1938; Hamby, 1942), the occurrence of bilateral intracavernous aneurysms is a rarity. This case may therefore be of interest, particularly as it appears to be the first in which the diagnosis was confirmed during life by angiography.

CASE REPORT

History.—G. C., a lady of 68 years, was admitted to the Neurosurgical Department of St. George's Hospital with the history that twenty months previously she had been suddenly stricken with severe generalized headache associated with vomiting. Two days later the left eyelid

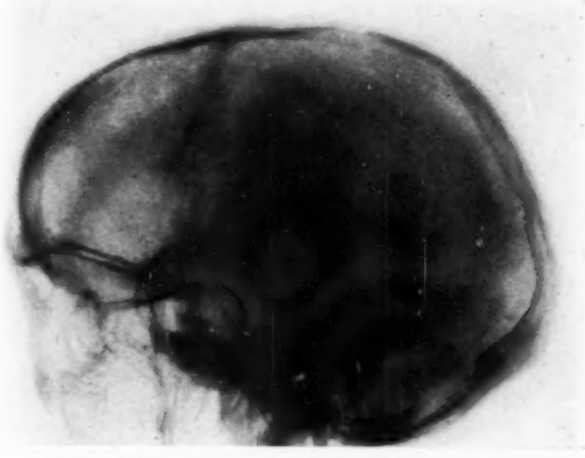

Fig. 265.—Radiograph showing elevation of the anterior clinoid processes and the ring of calcification.

drooped and an intense burning pain affected the left side of her forehead and face. When the eyelid was lifted up it was observed that the eyeball was immobile and she saw double. The condition persisted unchanged to the time of admission to hospital, when her main complaint was of the pain in her forehead.

She also explained that twenty years earlier, at the age of 48, she had experienced these identical symptoms of sudden headache and vomiting, followed on that occasion, however, by pain in the right side of her face and a total paralysis of the right eye, with a dropped eyelid. The eye had remained closed for eighteen months, after which recovery of ocular movements began and continued (she stated) during the ensuing seven years, leaving a residual weakness of abduction. The facial pain had cleared up in a few months.

On Examination.—Right eye: The pupil was small and reacted sluggishly to light and accommodation. Ocular movements were full except for the weakness of the external rectus muscle, abduction being limited by one half. Left eye: Complete ptosis. An internal and external ophthalmoplegia, which was total except for a few degrees only of abduction, adduction, and depression.

No sensory loss could be found over the left side of the forehead and face, despite careful testing, but the corneal reflex was diminished. Function of the muscles of mastication was unimpaired.

The right trigeminal field, motor, sensory, and reflex, was normal.

There was nothing further of note in the central nervous system. No intracranial bruit was audible. The pressure and constituents of the cerebrospinal fluid

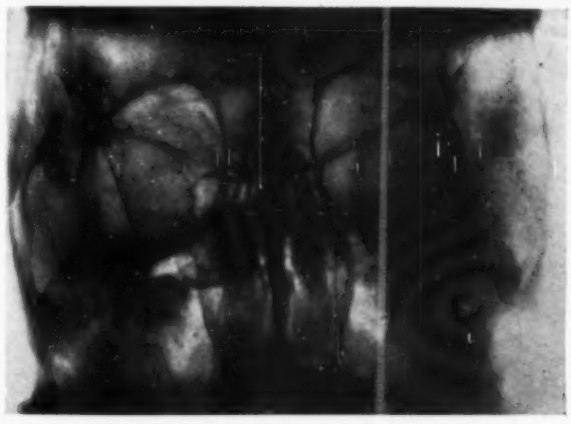

Fig. 266.—Radiograph of the orbits, showing expansion of the sphenoidal fissures.

were normal. Wassermann reaction in blood and cerebrospinal fluid was negative. Blood-pressure 142/84 mm. Hg.

X-ray examination of the skull (*Fig.* 265) showed 'sharpening' and elevation of the anterior clinoid processes, with destruction of the posterior processes. The

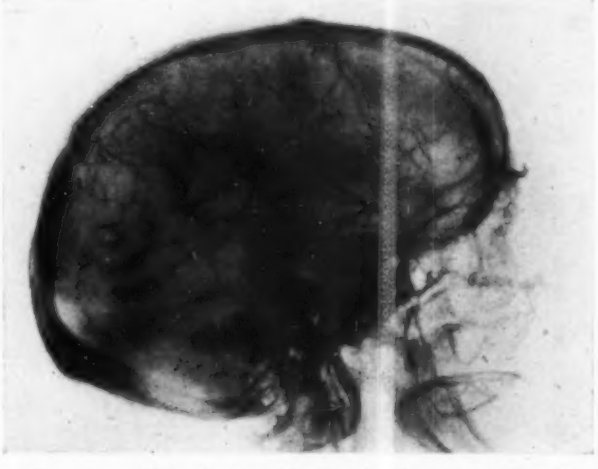

Fig. 267.—Right-sided arteriogram showing the aneurysmal sac and the contrast medium entering the upper half.

floor of the sella turcica was irregularly deepened. In the parasellar region on the right (the side of the first ophthalmoplegia) there was a ring of dense calcification measuring $2{\cdot}8 \times 2{\cdot}8 \times 3{\cdot}4$ cm. In the view taken of the orbits (*Fig.* 266) both sphenoidal fissures were seen to be expanded and the inferior margin of the lesser sphenoid wing on the left was eroded, with a resultant thin lateral wall to the optic foramen.

Ventriculography, which included good delineation of the temporal horns, disclosed no abnormality.

An arteriogram (using thorotrast) was performed on the right side and confirmed that the calcified ring formed

FIG. 268.—Anteroposterior view of right arteriogram.

the wall of a large saccular aneurysm of the internal carotid artery in the cavernous sinus, only the upper half of the sac filling with contrast, the remainder presumably being occupied by clot (*Figs.* 267, 268).

A further arteriogram on the left side (*Fig.* 269) revealed an aneurysm occupying the anterior part of the

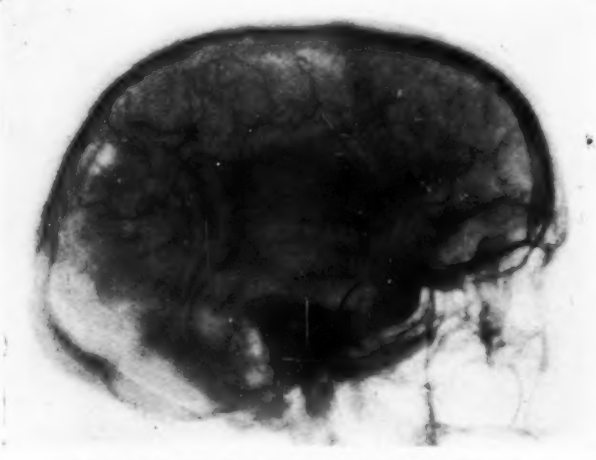

FIG. 269.—Lateral view of the left-sided aneurysm.

left cavernous sinus, possessing a more elongated sac, its apex rising above the anterior clinoid process.

TREATMENT.—The question of ligation of the left internal carotid artery in the neck was considered, in order to relieve the trigeminal pain, but it was thought at that time to be inadvisable in view of the patient's age and the condition of her arteries.

The patient was kept under observation for a further eighteen months and was re-admitted suffering from a severe senile dementia. The left facial pain had increased and was now associated with hypalgesia in all three divisions of the left trigeminus, but without motor involvement. There had been no recovery in the movements of the left eye.

The patient lapsed into coma and died a few days after admission.

AT AUTOPSY.—The brain showed uniform atrophy of the gyri and a rounded impression on the inner aspect of each temporal lobe corresponding to their contact with

FIG. 270.—Photograph of the base of the skull to show the aneurysmal sacs and their relationship to the oculomotor nerves.

the aneurysmal sacs. The two aneurysms enclosed in the dura of the cavernous sinuses can be seen in *Fig.* 270, with the relationship of the oculomotor nerves to them.

COMMENT

This case illustrates many of the clinical and radiographical features characteristic of aneurysms in this situation and emphasizes particularly the fact that they have little tendency to rupture into the subarachnoid space owing to the support given to the stretched arterial wall by the tough dural lining of the cavernous sinus. In aneurysms as large as these there is also little risk of the formation of a carotico-cavernous fistula owing to the complete obliteration of the venous channels of the sinus by the distended sac.

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CARCINOMA OF THE CYSTIC DUCT

By D. A. T. FARRAR, St. Bartholomew's Hospital

Primary carcinoma of the cystic duct is a rare condition. Very few cases have been reported in the literature and most of these cannot be regarded as proven. No satisfactory reports of the incidence of the condition appear to have been made, though it is mentioned in several series of post-mortem statistics (Reifferscheid, 1949).

The literature has been abstracted up to 1940 by Stewart, Lieber, and Morgan (1940), who found 27 cases reported. In a critical review they did not consider that any of these cases were "indisputable instances of primary carcinoma of the cystic duct". They divided the 27 cases into four groups:—

Group I—14 cases with gross neoplastic involvement of the gall-bladder.

Group II—7 cases in which the gall-bladder was not examined and in which there was no microscopical study of the growth.

Group III—5 cases in which the gall-bladder was not examined microscopically.

Group IV—1 case with multiple abdominal metastases, possibly a primary not in the cystic duct.

The cystic duct is a short structure. Far commoner sites for primary carcinoma are the gall-bladder and the hepatic and common bile-ducts which may easily invade the cystic duct. Before a final diagnosis of carcinoma of the cystic duct can be made, certain criteria must be satisfied:—

1. The growth must be restricted to the cystic duct.

2. There must be no neoplastic process in the gall-bladder, hepatic or common bile-ducts.

3. A histological examination of the growth must confirm the presence of carcinoma cells.

Since the paper by Stewart, Lieber, and Morgan, 5 more cases have been recorded (Braunschwig and Clark, 1941; Oppenheimer, 1942; Weimerhaus, 1941; Ciocatto, 1945; Lie Kian Joe, 1947) and one further case is reported here. Of the 5 cases, one (Lie Kian Joe, 1947) is not considered to be proven. It is similar to the case in Group IV above, with multiple metastases. The remaining 4, and the case here reported, appear to satisfy the above criteria and are accepted as cases of primary carcinoma of the cystic duct.

CASE REPORT

The patient, a male aged 66, was admitted to St. Bartholomew's Hospital on Dec. 24, 1949. He complained of pain under the right costal margin. During the night before admission he experienced severe pain in the right hypochondrium, typical of biliary colic. There was some nausea and he vomited once. There was no diarrhœa, the bowels were regular.

He gave a previous history of a gastric ulcer, diagnosed radiologically eight years before and treated medically. There had been an exacerbation three years before, treated medically. He occasionally suffered from mild attacks of indigestion with epigastric pain and flatulence. There had been no previous attacks of biliary colic.

On Examination.—He was seen to be distressed by severe pain. He was not jaundiced, his temperature was normal. The chest was emphysematous, with poor expansion and distant heart-sounds. The heart-sounds were normal and the B.P. 150/88. There was tenderness and rigidity in the right hypochondrium. Murphy's sign was positive. The urine did not contain bile and the stools were normal in colour. The following investigations were performed: hæmoglobin 86 per cent (Haldane); R.B.C. 5,000,000; W.B.C. 21,000; serum-bilirubin, 1 mg. per 100 c.c.; plain radiograph of abdomen, normal.

The pain rapidly settled after injections of morphine and atropine. It was thought that the biliary colic might be due to a stone in the cystic duct.

At Operation (Jan. 3, 1950).—Laparotomy was performed under spinal anæsthesia. The abdominal cavity

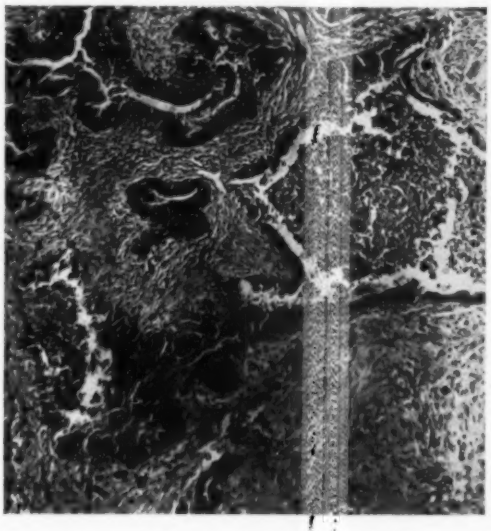

Fig. 271.—Section of the growth showing the alveolar formation and cells with dark-staining granular cytoplasm. (× 80.)

was opened through an upper right paramedian incision. The gall-bladder was found to be distended; the liver was not enlarged. A small lump, the size of a pea, thought to be an impacted stone, was palpated in the cystic duct about 1·25 cm. from its termination. The cystic duct was divided and ligated close to the junction with the hepatic duct. The cystic artery and vein were identified, ligated, and divided. The cystic duct, cystic gland, and gall-bladder were then removed. The cystic duct was opened at once and seen to contain a small, whitish, papillomatous growth. The gall-bladder bed was oversewn. The common duct was opened and explored, but there was no stone, obstruction, or evidence of new growth. A small rubber tube drain was laid in the common hepatic duct and the common bile-duct closed over it. Secondary deposits were not palpable in the liver or porta hepatis; no other abnormality was found. The wound was closed in layers, with a separate drain to the gall-bladder bed.

Progress.—The patient made an uninterrupted recovery. The drain in the common bile-duct was removed on the eleventh post-operative day after a choledochogram had shown a clear passage for fluid into the duodenum. He was discharged on the twenty-third day after operation.

He was seen six months later, when he had no abdominal symptoms and on examination did not show any signs of recurrence.

PATHOLOGY.—The specimen consisted of a gall-bladder containing dark thick bile and measuring 8 cm. in length and 3 cm. in diameter, together with 5 cm. of cystic duct. The wall of the gall-bladder showed some fibrous thickening. The mucosa had lost the normal fine folding and showed brown mottling in places. In the cystic duct 4 cm. from the gall-bladder was a papillomatous tumour 0·5 cm. in diameter, which was firmly attached to the wall of the duct and projecting into and apparently blocking the lumen. Microscopically the gall-bladder showed a subacute inflammatory reaction. The growth in the duct was a fairly well-differentiated adenocarcinoma with some alveolar formation. The cells were chiefly columnar and cubical, with dark-staining granular cytoplasm (*Fig.* 271).

DISCUSSION

The reported cases are too few in number to provide more than interesting points of similarity. In four of the five cases (*Table I*) the patient was a male, the single female patient being also the sole possessor of gall-stones. This sex incidence is similar to that found in carcinoma of other parts of the extrahepatic bile-ducts—70 per cent male, 30 per cent female (Stewart, Lieber, and Morgan, 1940).

The symptoms in two cases were painless jaundice, and in three cases pain in the right hypochondrium. In the former the tumour pressed upon the common bile-duct, giving rise to obstruction; in the latter the tumour obstructed the cystic duct. The physical signs were variable. In three cases the liver was enlarged on palpation, in two an enlarged gall-bladder was palpable; in two there was obstructive jaundice and in two cases there was tenderness under the right costal margin.

A correct pre-operative diagnosis was not possible. All the patients were submitted to laparotomy, when the condition was recognized (*Fig.* 272). In each case cholecystectomy and removal of the tumour was performed, the method of doing the latter varying. In *Case* 1 (Braunschwig and Clark) the gall-bladder was small and the cystic duct distended by growth. Cholecystectomy was performed and the cystic duct removed, together with a portion of the common bile-duct and surrounding tissues, *en bloc*. The hepatic artery, which was adherent to the growth, was injured and ligated. The common bile-duct was anastomosed over a T-tube (*Fig.* 272). In *Case* 2 (Oppenheimer) the tumour occupied the proximal end of the cystic duct. The gall-bladder, cystic duct, growth, a portion of the common bile-duct, and two lymph-nodes were all removed. The common bile-duct was anastomosed over a T-tube. In *Cases* 3, 4, and 5 (Weimerhaus, Ciocatto, and the author respectively) cholecystectomy was performed with removal of the cystic duct and tumour. In *Case* 5 the common duct was explored and drained.

There is no follow-up note on two of the cases, but they were discharged from hospital fit. Of the remaining three, *Case* 1, in which the hepatic artery had been tied at operation, died on the twenty-second post-operative day from liver failure. *Case* 2 died eleven months after operation, presumably from secondary deposits; it is interesting to note that the T-tube was not removed. *Case* 5 was alive and well six months after operation.

Table I.—DETAILS OF CASES REPORTED SINCE 1940

CASE	SEX	AGE	SYMPTOMS	SIGNS	GALL-BLADDER	COMMON BILE-DUCT	OPERATION	HISTOLOGY	PROGRESS
1. Braunschwig and Clark (1941)	M.	63	Painless jaundice	Liver not palpable. Icteric index 115. Radiography showed no abnormality	Small. Contained dark bile	Obstructed by pressure	Cholecystectomy, removal of tumour and a portion of common duct. Hepatic artery injured	Adenocarcinoma. Some alveolar formation. Gall-bladder normal	Died on 22nd day from liver failure. P.M. showed no metastases
2. Oppenheimer (1942)	M.	57	Painless jaundice	Enlarged liver palpable. Icteric index 81. Radiograph showed no abnormality	Distended with white bile	Obstructed by pressure	Cholecystectomy, removal of tumour and a portion of common duct. T-tube left in	Adenocarcinoma. Gall-bladder normal	Died 11 months after operation, ? from secondary deposits, no P.M.
3. Weimerhaus (1941)	M.	71	Pain in the right hypochondrium. Abdominal mass	Liver and gall-bladder enlarged and palpable. Radiograph showed no abnormality	Distended	No obstruction	Cholecystectomy with removal of the cystic duct and tumour	Adenocarcinoma. Gall-bladder normal	Discharged fit
4. Ciocatto (1945)	F.	50	Pain in the right hypochondrium	Palpable mass in the area of the gall-bladder. Tender. Radiograph showed gall-stones	Distended. Contained stones	No obstruction	Cholecystectomy with removal of the cystic duct and tumour	Adenocarcinoma. Chronic cholecystitis	Discharged fit
5. Author's case	M.	66	Pain in the right hypochondrium, biliary colic	Enlarged liver just palpable. Tenderness in the right hypochondrium. Radiograph showed no abnormality	Distended, dark bile	No obstruction	Cholecystectomy with removal of the cystic duct and tumour	Adenocarcinoma. Subacute cholecystitis	No signs of recurrence after 6 months

Macroscopically, growths of the extrahepatic bile-ducts have been divided into two types by Bolli (1946) —the localized and the diffuse (*Table II*). Of the five cases, *Cases* 1 and 2 were of the diffuse type,

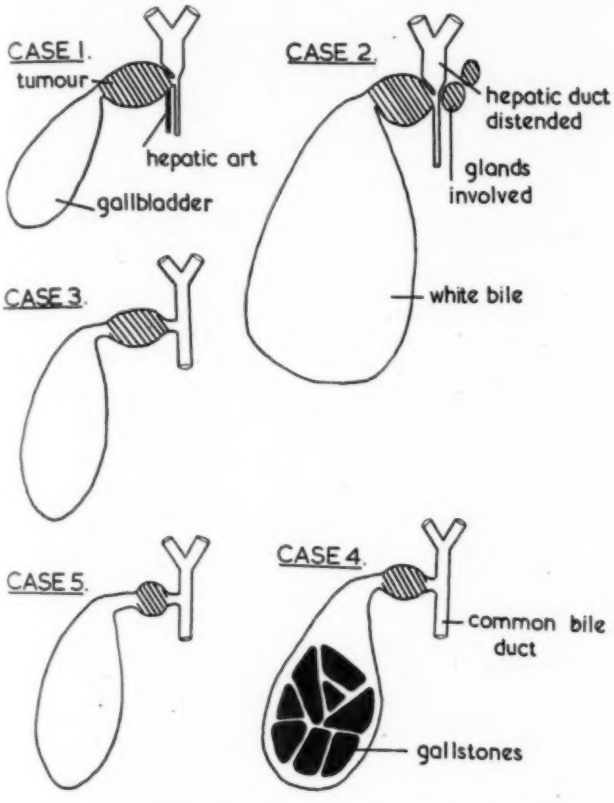

FIG. 272.—Diagrammatic representation of the findings at operation in the cases described.

Case 3 appears to have been a localized annular type, and *Case* 5 a localized villous type. Histologically all the tumours were adenocarcinomata. The cells showed some alveolar formation and in three cases were said to have a granular cytoplasm. The histology of these growths was essentially similar

Table II.—TYPES OF CARCINOMA OF THE BILE-DUCTS (*After Bolli*, 1946.)

A. DIFFUSE :—
Partial. Affecting a portion of the wall.
Complete. Encircling the duct wall.

B. LOCALIZED :—
Villous.
Vegetative.
Nodular.
Pedunculated.
En plaque.
Annular.

to that described in carcinomata of other extrahepatic bile-ducts (Stewart and others, 1940; Renshaw, 1922).

SUMMARY

A case of primary carcinoma of the cystic duct is described. Only 4 other certain cases of this condition appear to have been recorded in the literature; these are reported. The 5 cases are compared and discussed.

I wish to express my thanks to Mr. Rupert Corbett, who performed the operation on the case here described, for his permission to publish this case and for his advice and encouragement. I am also indebted to Dr. R. Cureton for the pathological report.

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PERITONEAL LOOSE BODY CAUSING ACUTE RETENTION OF URINE

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PERITONEAL loose bodies are rare, and the complication of acute retention due to impaction in the rectovesical pouch is probably unique.

CASE REPORT

A deaf old man, aged 79, was admitted as an acute surgical emergency with sudden retention of urine. This had happened a few hours previously without any premonitory symptoms of straining or other difficulty. For months he had had a severe productive cough and his acute bronchitis and emphysema were thought by the casualty officer to render him unsuitable for surgery. A catheter was tied into the urethra and he was admitted to the ward with the diagnosis of acute prostatic obstruction. Examined next day this rather frail chesty old man was found to have nothing abnormal in the abdomen except for a healed appendicectomy scar. The catheter was draining well. On rectal examination a peculiar state was encountered. The prostate was symmetrical and slightly enlarged; it was of soft consistency. Above the prostate and slightly to the left side was felt a very hard almost stony mass which on palpation was estimated to be about one inch in diameter. At first this mass was thought to be completely fixed but on putting the patient into the knee-elbow position the mass could be manipulated forward a short distance. It was considered to be a calculus either in a diverticulum of the bladder, or in the lower left ureter. Radiological investigations revealed an irregular calcified area slightly to the left side of the bladder (*Fig.* 273). An intravenous pyelogram showed good renal function on both sides but a marked bilateral hydronephrosis and hydro-ureter (*Fig.* 274). The urine was sterile but despite this finding the diagnosis of vesical calculus, probably in a diverticulum,

was maintained. The chest condition was much improved after a week of postural drainage and antibiotic therapy. Operation was advised.

OPERATION.—The bladder was exposed through a midline sub-umbilical incision. The bladder was opened and although slightly hypertrophied proved to be otherwise normal. No calculus or diverticulum was found. The prostate was slightly enlarged but did not appear to be obstructing the urethra. The trigonal region was pushed forward as if stretched over a mass lying extravesically. The hard lump felt rectally proved by palpation through the posterior bladder wall to be much larger than originally supposed. It remained fixed despite bimanual pressure from the rectum and the bladder. The left lateral wall of the bladder was freed externally and a finger passed posteriorly. Suddenly by manipulation from this angle the mass shot away upwards out of reach. It was assumed to have escaped into the general peritoneal cavity and was

FIG. 273.—Plain film of pelvic area showing opacity to left of indwelling catheter. Irregular calcification is shown.

immediately pursued by opening the peritoneum above the bladder reflection. The free object illustrated in *Fig.* 275 was easily located. The pelvis was explored but no other loose bodies were felt. The peritoneum was closed. The bladder was closed and an indwelling urethral catheter left in position. Convalescence was uninterrupted. Some difficulty in micturition continued and an intravenous pyelogram three weeks after operation showed the relief of obstruction of the ureters.

THE SPECIMEN.—The size and surface appearance are well shown in the illustrations (*Figs.* 275, 276). The consistency is peculiar as if there is an elastic covering over a hard central core. The radiological appearance of the isolated specimen corresponds with the opacity on the plain film taken on admission. On section the tumour cuts with difficulty with a knife and in the centre there is a small core about 2 mm. in diameter and as hard as flint. Surrounding this core are laminations of rather tough elastic tissue with cheesy layers intervening. These layers are structureless on histological examination and are composed of fibrinous material with a variable deposition of calcium salts. The small central core defies analysis and its shape and appearance are indeterminate.

DISCUSSION

That this solitary loose body determined an acute retention of urine is certain : the origin of the loose body is unknown. Ross and McQueen (1948) found a very similar mass in a man of 69. A lump was felt in the upper abdomen and, as the diagnosis was uncertain, laparotomy was done. The lump was found to be a smooth white loose body the size of a hen's egg lying in the lesser sac between the stomach and colon. It was easily removed. A subsequent radiograph revealed a central opaque core very like that described in the present case. Hunt (1919) collected records of eleven cases of peritoneal loose bodies, usually multiple. Small loose bodies are seldom recorded but quite often found in hernial sacs. Usually, as in the case described by McIntosh

FIG. 274.—An intravenous pyelogram shows good function. There is gross dilatation of the pelves and ureters of both kidneys.

to Ross and McQueen (1948), peritoneal loose bodies are found incidental to operation or autopsy. The origin of such masses is uncertain.

Recognizable " foreign " bodies which have entered the peritoneal cavity from the gut or from external sources are in a different category. The term " loose " body usually implies a derivation from the separation of tissue from the lining of the peritoneal cavity. It may be regarded as analogous to the loose body occurring in a joint. Detached organs or other anatomical structures may be included under the title. Thus Harrigan (1917) and Hunt (1919) believe that the appendices epiploicæ may undergo torsion following inflammation and detach to lie free in the peritoneal cavity. There they may increase in size by the deposition of fibrin. Johnstone (1927) performed a laparotomy, having diagnosed a radio-opaque movable mass in the pouch of Douglas. He found a free calcareous mass about

the size of a Brazil nut. On section, the ghost of ovarian epithelium was recognized. There are a few other records of loose bodies derived from pedunculated subperitoneal fibroids or from ovarian tumours. In the female old blood-clot from a tubal

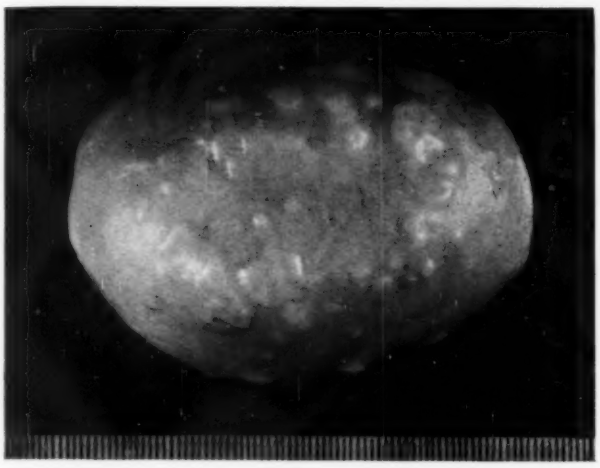

FIG. 275.—The glistening rather knobbly surface is shown. The scale is in millimetres and the lump is about 7 cm. in its long axis and about 5½ cm. in its transverse diameter.

abortion has been considered the possible source of large fibrinoid tumours lying free in the pouch of Douglas. I am indebted to Professor Wells for a brief note of a case of this type.

A woman of middle age was operated upon for an acute abdominal emergency. On palpating the pelvis the loose body shown in *Fig.* 277 was lifted out of the

FIG. 276.—The cut surface shows the laminated character of the mass. In the centre of one half is shown the minute flint-like core.

recto-uterine pouch. It was lying quite free and in appearance was lobulated and smooth and of reddish brown colour. It was thought to be composed of organized blood-clot derived from a hæmorrhage probably tubal, which had happened months or years before. The loose body had no relation to the acute condition.

Mr. Clifford Morson showed a peritoneal loose body the size of a small orange at a meeting of the R.S.M. in 1927. This was found at autopsy in the peritoneal cavity of an old man who died in uræmia after an operation for acute retention of urine. It lay in the rectovesical pouch but was not impacted.

In the present instance the loose body occurred in an elderly man and is not easily explained. It is known that an appendicectomy had been performed and it is possible that some foreign material entered the peritoneal cavity and formed the centre for fibrin deposition in the course of a prolonged low-grade peritoneal infection. One may speculate on the nature of the escaped foreign material—a fragment of fæcolith would appear to be the most likely material.

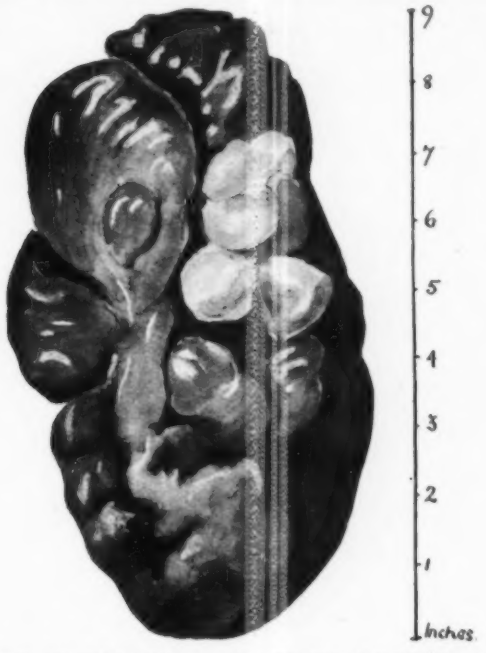

FIG. 277.—Reproduction of a water-colour sketch made by Professor Wells after the removal of a large loose body from the pouch of Douglas of a woman about 30 years old. (× ½.)

Reference may be made in conclusion to the possibility of loose bodies following the injection of liquid paraffin into the peritoneal cavity—a procedure once advocated in the vain hope that it might prevent adhesion formation (Cruickshank, 1941).

SUMMARY

Peritoneal loose bodies are rare. Symptoms are unusual and the singular occurrence of acute urinary retention from impaction of a large loose body in the rectovesical pouch is described.

My thanks are due to Professor Wells for permission to record this patient treated in his unit at Broadgreen Hospital and for allowing me to include his own case. Mr. Clifford Morson has kindly given me a note regarding the loose body he demonstrated in 1927. Miss Barbara Duckworth prepared the reproduction of Professor Wells's original water-colour sketch. Mr. Lee, of the Liverpool University Photographic Department, prepared prints of my own case.

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HÆMOPERITONEUM DUE TO RUPTURE OF A SEMINOMA IN AN ABDOMINAL TESTIS

By G. GORDON CROWE, Stoke-on-Trent

Hæmoperitoneum due to rupture of a seminoma in an abdominal testis is very rare and has never before been described in the British literature. French surgeons, however, have described this occurrence on three occasions.

Arousseau in 1926 describes 76 cases of malignant tumours arising in an abdominal testis, and in only 1 of these cases had rupture occurred. This was a case attributed to Lecène in 1913 which happened in a man of 40 years who had no testicle on the right side of the scrotum. He was suddenly stricken with abdominal pain and nausea and he gave the impression of having an intra-abdominal hæmorrhage. The diagnosis of torsion of an abdominal testis was made. A painful abdominal tumour was felt and at laparotomy blood was found in the peritoneum, and a bleeding fissured tumour twisted on its pedicle was removed. Section proved it to be a seminoma. The patient lived for eleven years and one month after operation.

Guibal and Goepfert in 1946 have described another case. This patient was a man of 42, who had been operated on for bilateral undescended testes at the age of 20. At this operation no testicles were found. This patient was diagnosed as either an appendicitis or a perforation, but at laparotomy he was found to have a hæmoperitoneum due to rupture of an abdominal seminoma which was attached to the posterior abdominal wall by a short twisted pedicle. He was treated by ligature of the pedicle and removal of the seminoma.

Bergeret has also described a case on which he operated in 1938 in a man of 40 who had bilateral undescended testes. At operation a pint of blood was found in the peritoneal cavity, and behind the cæcum was found an ectopic right testicle about four times the size of a normal testicle and bleeding from its lower pole. He was treated by section of the pedicle and removal of the tumour. This was proved to be a seminoma.

CASE REPORT

P. B., aged 29 years, was a professional footballer who had been operated on in 1940, while in the Forces, for an undescended testicle and inguinal hernia on the right side. He was admitted to hospital on August 10, 1950, complaining of the onset of sudden stabbing abdominal pain eight hours previously.

The pain began in the right iliac fossa and spread over the whole of the lower abdomen. He also complained of pain in both shoulders. His bowels had opened before the pain started and he had passed flatus since the onset of the pain. He had vomited once after the onset of the pain.

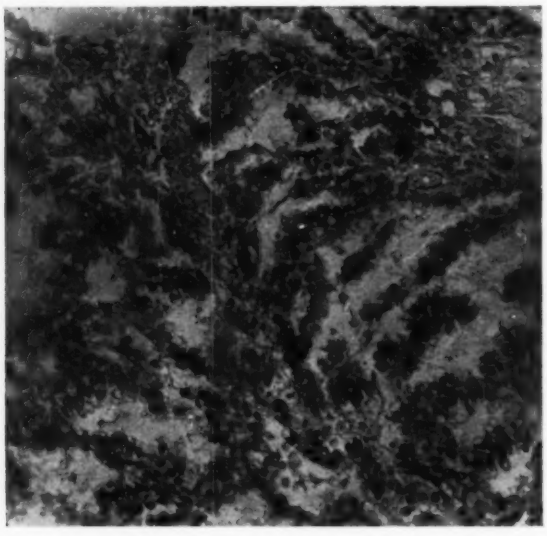

Fig. 278.—Pseudopapilliform arrangement of testicular carcinoma. (× 128.)

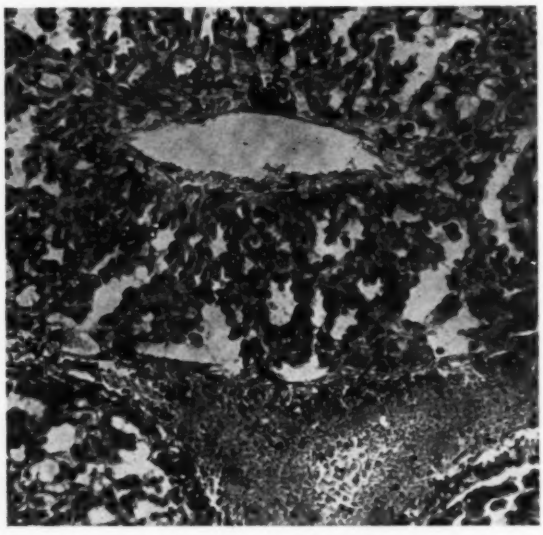

Fig. 279.—A different portion of the same tumour growing around a vessel. (× 128.)

On examination he appeared pale, rather restless, and was sweating. His temperature was 97·6°, pulse 76, and his respirations were 20. His abdomen was moving with respiration and there appeared to be slight distension of his lower abdomen. There was increased tone in the right iliac fossa, but no generalized rigidity. He was slightly tender in his lower abdomen and this was most marked below and to the left of the umbilicus. Pressure in his right iliac fossa caused a dull ache in his shoulders. Liver dullness was not impaired and there were no bowel signs present on auscultation. After four hours the pain in his shoulders had increased and there was a slight rise in his pulse-rate. It was felt that he had a hæmoperitoneum and laparotomy was decided upon. He had a right inguinal operation scar and no testis was present in the scrotum on the right side.

His abdomen was opened through a right paramedian incision and about three pints of blood were found in the peritoneal cavity. This was found to arise from a

tumour about the size of an orange which was attached by a pedicle to the posterior abdominal wall at the level of the common iliac bifurcation. This tumour was bleeding from a fissure of its lower part. The pedicle was clamped and the tumour was removed. The abdomen was closed in layers.

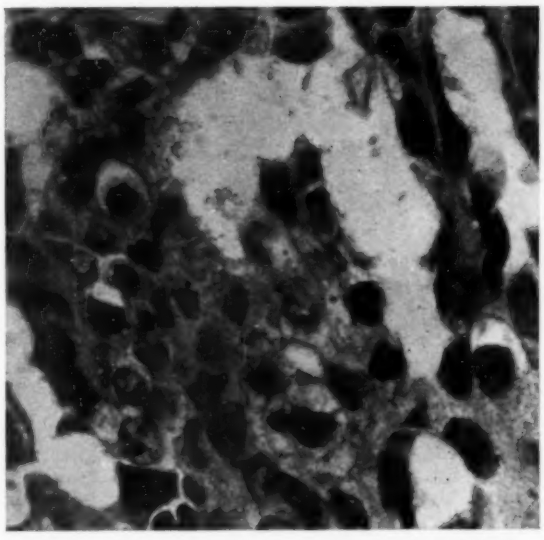

FIG. 280.—Portion of *Fig.* 278 showing cytological arrangement including several mitoses. (× 320.)

The patient made an uneventful recovery and was discharged from hospital twelve days later. He is at present having a course of deep X-ray therapy. Section of the tumour showed an anaplastic carcinoma which had the appearance of an intra-tubular carcinoma of the testis (*Figs.* 278–280).

The operation notes show that in December, 1940, while he was in the Forces, he had a right inguinal hernia repaired and the testicle appeared to be completely atrophied. They do not state whether the testicle was indeed removed at that time.

The problem in this case is to correlate his operation of 10 years previously with the ruptured seminoma found on this occasion. Was the tumour found at operation arising in an abdominal testis, or was the testis removed at operation when his hernia was dealt with 10 years previously, the tumour found at laparotomy being a secondary spread?

We know that the prognosis of malignancy in the retained testicle is much worse than tumours of the normal organ. Although in 1938 Gordon-Taylor reported three cases where life was prolonged remarkably after removal of an inguinal seminoma, this he attributes to the X-ray therapy which was given to these cases after operation. One case was alive and well 16 years after simple orchidectomy for a seminoma in an imperfectly descended testicle just outside the external abdominal ring. In the second case the patient was alive and well 10 years after removal of a teratoma in an inguinal testis, and the third patient was killed in an accident 12 years after simple orchidectomy for a seminoma in an inguinal testis. Each of these cases was given X-ray treatment following operation.

In the pre-radiation days, however, Kocher found only one case of cure in a collected series of 55 cases of malignant disease of a retained testis. In Bulkeley's series only one was alive after two years. In Odiorne and Simmons' (1904) series of 6 cases all except one were dead within a year, and Chevassu (1906) found but one survival of longer than 18 months.

These findings lead to the belief that in the present case it is extremely unlikely that the tumour found at operation was a secondary, and that at his original operation 10 years before his testis could not have been removed, but was placed in the abdomen or was left in situ and subsequently a seminoma developed in the abdominal position found at operation.

Two cases have been reported of malignant disease supervening in a testicle replaced by the operator inside the abdomen. Gordon-Taylor (1938) reported one case where the patient at the age of 15 years was said to have had a left undescended testicle returned inside the abdomen. Six years after the operation this patient appeared with a large fixed tumour in the left iliac fossa, just inside the internal abdominal ring. Metastases soon appeared in the thorax and in the left side of the neck and grew at a phenomenal rate. Death quickly supervened.

Romiti (1923) recorded a similar case when an inguinal retained testis was replaced inside the abdomen when a left inguinal hernia was repaired in a man of 37. Malignant disease subsequently developed in the testis.

I wish to thank Dr. C. Giles for the Pathological report and Dr. E. M. Stirk for the photomicrographs.

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REVIEWS AND NOTICES OF BOOKS

Plasma Clot Suture of Peripheral Nerves and Nerve Roots. Rationale and Treatment. By I. M. TARLOV, M.D., Associate Professor of Neurosurgery, New York Medical College, New York City. 6 × 9 in. Pp. 116 + xii, with 47 illustrations. 1950. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 40s.

THIS book reviews the experimental work upon which the practice of plasma clot suture of nerves is based. It provides full details of the comparative value of different varieties of plasma for this purpose, and of the technique employed by the author in his own researches upon animals. Frequent reference is made to the technique of nerve suture, including plasma clot suture, in man, but the author has nothing of his own to add to the experience of other surgeons here and in the United States whose contributions to the surgery of the peripheral nerves and the spinal nerve-roots are already well known. Its chief value, therefore, must be as a reference book for those engaged in neurosurgical research.

Indications for and Results of Splenectomy. By FREDERICK A. COLLER, M.D., ALEXANDER BLAIN, III., M.D., and GOULD ANDREWS, M.D., from the Departments of Surgery and Medicine, University of Michigan Medical School, Ann Arbor, Michigan. 5½ × 8½ in. Pp. 100 + viii, with 16 illustrations. 1951. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 16s. 6d.

IT is only to be expected that the booklets in this series of American Lectures in Surgery, of which this on Splenectomy is one, would vary in their usefulness. Some are mere statements of current teaching, whereas others combine this with records of personal experience which greatly enhance their value. The authors of this little book on the surgery of the spleen, Drs. Coller, Blain, and Andrews, have followed the latter course, and have given their readers the benefit of their own experience, carefully documented and with thoughtful, well-informed commentaries. The result is a gem of surgical literature which compresses admirably into its 100 pages all that a general surgeon needs to know about the spleen, and contains a short bibliography for the guidance of those who are seeking for further details. The style is clear and concise, and the authors deserve much credit for making such good use of limited space; there are no redundancies, no waste of words, yet everything seems to be mentioned. It is not too much to say that if this were the summary of a monograph on the spleen, the major work would take its place at once among the classics of medical literature.

Instruments and Apparatus in Orthopædic Surgery. By E. T. NANGLE, M.B., Ch.B., F.R.C.S., formerly Resident Surgical Officer, Royal National Orthopædic Hospital, Stanmore, and Wingfield-Morris Orthopædic Hospital, Oxford. With a Preface by H. J. SEDDON and JOSEPH TRUETA. 7⅞ × 9¾ in. Pp. 231 + xv, with 200 illustrations. 1951. Oxford: Blackwell Scientific Publications. 42s.

AT first sight it might be thought that there was no need for such a book as this, but on reading the volume one is convinced that there is a very real necessity for it.

The surgeon of to-day has at his disposal a bewildering variety of instruments and appliances for the correction, treatment, and support of diseases of the skeleton and disorders of the locomotor system. All surgeons who deal with traumatic lesions should not only have a knowledge of traumatic surgery but should also have a sound knowledge of 'brace-making' therapeutics.

There is little in surgical literature concerning the construction of appliances, and many a young surgeon has had to be content with an instrument-maker's catalogue.

Mr. Nangle has written and illustrated a first-class book, which has the great advantage that the author writes about apparatus that he has used and tested and found to be of real value to the orthopædic surgeon.

Syllabus of Human Neoplasms. By R. M. MULLIGAN, M.D., Professor of Pathology in the University of Colorado School of Medicine. 7 × 10 in. Pp. 317, with 230 illustrations. 1951. London: Henry Kimpton. 52s. 6d.

THIS book is what its author has called it, namely, a syllabus or précis of human neoplasms. It contains the pertinent pathological facts about the more common neoplasms together with a correlation of their salient clinical features. The photomicrographs, which are numerous, are excellent and give the reader a good idea of the histological processes.

This book should be of value to the senior student and the resident medical officer who require a ready reference volume to neoplastic pathology.

Rest and Pain. By JOHN HILTON, F.R.S., F.R.C.S. Edited by E. W. WALLS, M.D., Ch.B., B.Sc., Professor of Anatomy in the University of London at Middlesex Hospital Medical School; and ELLIOT E. PHILIPP, M.A., M.B., B.Chir., M.R.C.S., M.R.C.O.G., Honorary Demonstrator in Anatomy at Middlesex Hospital Medical School; in collaboration with H. J. B. ATKINS, D.M.M.Ch., F.R.C.S., Director of the Department of Surgery, Guy's Hospital. 5½ × 8½ in. Pp. 503 + xxxvi, with 105 illustrations. 1950. London: G. Bell & Sons Ltd. 25s.

MANY a student's first impression of *Rest and Pain* must have been that, wonderful though it was, it might have been much more valuable to him if it had been interpreted in the language of modern physiology and pathology. This the present editors have attempted to do by means of appendices to every Lecture which, without detracting one whit from the grandeur of the original work, have added enormously to its usefulness as a student's text-book. Ever since the Lectures were first published they have been read by many medical undergraduates and post-graduates, but there is no doubt that they ought to have been read by many more; and this clear presentation of the classic work should provide the material and the stimulus for a very greatly increased range of readers. The present edition is embellished by an Introduction, written by Mr. Hedley Atkins, which gives an account of Hilton's life and refers briefly to the influence which he exerted upon surgical thought in his own day; and also by biographical notes on many of the physicians and surgeons to whom Hilton referred in the course of his Lectures. In the Preface to the first edition John Hilton stated that although he had wished to revise and remodel the Lectures before they were published, the urgent solicitations of his friends for their early appearance prevented his doing so. Though the Lectures themselves have never been altered, except for a few words, Hilton's original intention has been fulfilled, in the earlier editions in the copious notes made by his disciple

Jacobson, and in this sixth edition in the appendices which are commentaries worthy of the original work, and and worthy also to rank with Jacobson's contributions to the same end.

Physical Diagnosis. By RAYMOND W. BRUST, M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania Medical School. Pp. 294 + xii. 1951. New York: Appleton-Century-Crofts, Inc.

IT is stimulating to-day to find a book devoted to physical diagnosis because the modern student is not taught physical signs as he should be, but is being taught by a series of tests or investigations.

Dr. Schnabel, who writes a Foreword to this book, rightly remarks: "Physical diagnosis coupled with the patient's story constitutes the keystone to successful medical practice."

In this book a very practical description of physical signs is given together with some excellent photographs which illustrate the most important physical examinations.

There are no X-ray photographs, and so there is no tendency towards the false positives or the misleading normals or the film that killed the patient.

This is a book that should be widely read not only by students but also by practitioners.

The 1950 Year Book of Urology. Edited by WILLIAM WALLACE SCOTT, M.D., Ph.D., Director, James Buchanan Brady Urological Institute; The Johns Hopkins Hospital; Urologist in Charge, The Johns Hopkins Hospital; Professor of Urology, The Johns Hopkins University School of Medicine. 5 × 7¼ in. Pp. 416, with 87 illustrations. Chicago, Ill.: Year Book Publishers Inc. $5.00.

THE appearance of this book under its new editor, Dr. W. W. Scott, marks a fresh place in its history. The editor himself contributes an introductory chapter on the advances in urology during the past decade and this alone provides an effective reply to those who decry the increasing tendency to specialization in this branch of medicine and surgery.

The abstracts of current literature are not too brief to be readable; they are well chosen and we hope that the difficulties which prevent more British articles being included will be surmounted. The annual appearance of this volume is eagerly awaited by every urologist; we extend to Dr. Scott every good wish for its continued success.

1950 Year Book of Neurology, Psychiatry and Neurosurgery. Edited by ROLAND P. MACKAY, M.D., Professor of Neurology, University of Illinois (Neurology); NOLAN D. C. LEWIS, M.D., Director N.Y. State Psychiatric Institute and Hospital (Psychiatry); and PERCIVAL BAILEY, M.D., Professor of Neurology and Neurological Surgery, University of Illinois. 5 × 7⅝ in. Pp. 627, with 121 illustrations. 1951. Chicago: Year Book Publishers Inc. $5.00.

EACH of the three sections of neurology, psychiatry, and neurosurgery is introduced by an account of a decade of progress in the subject. In neurology, R. P. Mackay stresses the advances which have been made in neurophysiology, diagnostic methods, the pathogenesis of diseases of the nervous system, therapeutic methods, and rehabilitation. In his introduction to psychiatry, N.D.C. Lewis chooses the biochemistry of metabolism in the cerebral nervous system as an example, but admits the limitations of our present knowledge. He finds serious attempts everywhere being made to improve this branch of medicine and believes that the most important advances in the future will be unexpected. In his introductory article on a decade of neurosurgery, Percival Bailey reminds us that Chipault, of Paris, developed this speciality in the nineties of the last century. Most of the great advances in it had been made before 1940 and the recent war has contributed little but advances in the treatment of infections. If further demonstration were needed, it has again been shown that bone is the material of choice for closing skull defects. In the past decade there have been technical advances such as new hæmostatic agents like fibrin foam and film, and gel foam and the short-circuiting operation of Torkildson in hydrocephalus. Surgery of the sympathetic system seems to have passed another peak and to be on the decline. These introductions make interesting reading to the accounts which follow of the well-chosen papers reviewed by the three editors, in their various sections.

Surgical Nursing and After-treatment. By H. C. RUTHERFORD DARLING, M.D., M.S., F.R.C.S., F.R.F.P.S. Glasg., Surgeon, Prince Henry Hospital and New South Wales Masonic Hospital, Sydney, N.S.W., and T. EDWARD WILSON, M.D., M.S., M.Sc., F.R.C.S., F.A.C.S., F.R.A.C.S., Assistant Surgeon, The St. George Hospital, Sydney. Tenth edition. 4¾ × 7¼ in. Pp. 630 + x, with 188 illustrations. 1951. London: J. & A. Churchill Ltd. 16s.

"SURGICAL nursing", as the senior author tells us, "is best learnt by actual experience in a well-equipped and up-to-date general hospital", but here is a book to which the nurse may turn for explanations of terms or phenomena which may have puzzled her. To make an ill patient comfortable, however, is a more important task for a nurse than to differentiate gastric from duodenal ulcer! Moynihan's tube is now of only historical interest, and perhaps the same may be said of certain of the operations shown for gastric ulcer, and need the nurse know these technicalities? It is curious to find no mention of Higginson's syringe.

There is much in this book which is admirable—too much, however, for the tired student nurse to master, and we hope that she will use it as a work of reference rather than a handbook to be read from cover to cover.

Primary Carcinoma of the Liver. A study in Incidence, Clinical Manifestations, Pathology and Aetiology. By CHARLES BERMAN, M.D., B.Ch. Rand. Senior Medical Officer, Consolidated Main Reef Mines and Estate Ltd., Maraisburg, Transvaal, S.A. With a Foreword by SIR ERNEST KENNAWAY. 7¼ × 9¾ in. Pp. 164 + xvi, with 83 illustrations. 1951. London: H. K. Lewis & Co. Ltd. 35s.

IT is given to few medical men to see many cases of primary carcinoma of the liver as the incidence is so small except in some areas, such as that of the Bantu of South Africa. It is interesting to note that primary carcinoma of the liver is the most frequent form of malignancy encountered in Bantu males. It was responsible for 88·6 per cent of all cancers in native mineworkers on the Witwatersrand, and for 31·9 per cent of cancers in Bantu males treated at the Johannesburg Non-European Hospital. These are remarkable figures, and Dr. Berman has put the medical profession of the world in his debt in writing this important monograph. To have investigated the clinical and pathological material of no fewer than 75 cases of primary carcinoma of the liver is no mean task. Dr. Berman's book, which is the outcome of these investigations and is the result of many years' hard work, will be read by all clinical observers and especially by pathologists in this country. This book is exactly what a monograph on a particular subject should be—namely, comprehensive, detailed, well-illustrated, and with a complete bibliography of published works on the subject.

We could do with more books of this calibre.

Acute Head Injury. By JOSEPH P. EVANS, M.D., Ph.D., Associate Professor of Surgery, University of Cincinnati College of Medicine, Ohio. Pp. 116 + v. 1951. Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 16s. 6d.

THIS small monograph of just over a hundred pages gives a concise description of the diagnosis and treatment of acute head injuries. It is divided into four definite parts: scalp injuries, skull fractures, meningeal hæmorrhage in its various forms, and cerebral trauma.

As the incidence of head injury in this mechanical age is so high, it behoves the profession to understand the rational principles of treatment of acute cranial and intracranial injury. This little monograph goes a long way to provide a lucid description of the treatment required for such injuries.

Diseases of the Heart and Circulation. By PAUL WOOD, O.B.E., M.D., Director, Institute of Cardiology, London; Physician, National Heart Hospital; etc. 6 × 9⅜ in. Pp. 589 + xxx, with 354 illustrations. 1950. "The Practitioner" Series. London: Eyre & Spottiswoode (Publishers) Ltd. 70s.

THE recent advances in many aspects of cardiology make this new text-book by such a well-known and able cardiologist as Dr. Paul Wood very welcome. Especially important is the fact that Dr. Wood has himself made numerous contributions to many of the subjects he discusses. His personal experience with cardiac catheterization is noteworthy in this respect.

The text is informative and readable; the material both clinical and non-clinical, is well presented, the many electrocardiograms are excellently produced and admirably expounded. In contrast, the chest radiographs are for the most part badly reproduced and some are quite useless, e.g., *Figs.* 20.02 and 20.03 in which bullets are supposed to be shown within the heart. No doubt in the next edition this defect will be corrected; it is certain that a second edition will soon be needed for such a sound and authoritative exposition of such an important subject. The chapter on injuries to the heart might also, with advantage, be made somewhat fuller. The principles and results of surgical treatment of arteriovenous aneurysm should also be more accurately and critically described and presented.

The Normal Encephalogram. By LEO M. DAVIDOFF, M.D., Director of Neurological Surgery, Beth Israel Hospital, New York City; and CORNELIUS G. DYKE, M.D., Late Associate Professor of Radiology, Columbia University. Third edition, revised by LEO M. DAVIDOFF. 6 × 9 in. Pp. 240, with 156 illustrations. 1951. London: Henry Kimpton. 42s.

THE third edition of this monograph has been very thoroughly revised and brought up to date by Dr. Davidoff and many new illustrations have been added. The radiographs illustrating the normal ventricular system of the brain are excellent.

Neurosurgeons to-day depend to a large extent on their radiological colleagues to help them in the diagnosis and localization of certain cerebral tumours. It is impossible to diagnose the abnormal if a thorough understanding of the normal does not form a sound background.

This book is a most useful volume not only to the neurosurgeon but also to the anatomist and neurologist. A very complete bibliography is found at the end of the volume.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

The Mode of Action of Anæsthetics. By T. A. B. HARRIS, M.B., B.S., D.A., F.F.A.R.C.S., Anæsthetist to Guy's Hospital and to St. Peter's Hospital for Stone. 5½ × 8½ in. Pp. 768 + xii, with 22 illustrations. 1951. Edinburgh: E. & S. Livingstone. 42s.

Surgical Forum. Proceedings of the Forum Sessions Thirty-sixth Clinical Congress of the American College of Surgeons, Boston, Massachusetts, October, 1950. 6¼ × 9¾ in. Pp. 665 + xlviii, with 260 illustrations. 1951. Philadelphia and London: W. B. Saunders Co. 50s.

Roentgen Manifestations of Pancreatic Disease. By MAXWELL HERBERT POPPEL, M.D., F.A.C.R., Associate Professor of Clinical Radiology, New York University. 6¾ × 9⅝ in. Pp. 389 + xiv, with 166 illustrations. 1951. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 77s. 6d.

Anæsthesia in General Practice. By STUART C. CULLEN, M.D., Iowa. Third edition. 5½ × 8 in. Pp. 292, with 36 illustrations. 1951. Chicago: Year Book Publishers Inc. $4.50.

Post-Graduate Lectures on Orthopedic Diagnosis and Indications. By ARTHUR STEINDLER, M.D., F.A.C.S., Professor of Orthopedic Surgery, State University of Iowa, Iowa City, Iowa. Volume II. Section A, Paralytic Disabilities; Section B, Static Disabilities. 6½ × 10 in. Pp. 198 + xiv, with 140 illustrations. 1951. Springfield, Ill.: C. C. Thomas. (Oxford: Blackwell Scientific Publications.) 45s.

Anatomy in Surgery. By PHILIP THOREK, M.D., F.A.C.S., F.I.C.S., Assistant Clinical Professor of Surgery, University of Illinois College of Medicine. 7 × 10 in. Pp. 970 + xxiv, with 720 illustrations, 211 in colour. 1951. Philadelphia and London: J. B. Lippincott Co. £9.